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Gender Differences in the Life Course Origins of Adult Functioning and Mortality

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Gender Differences in the Life Course Origins of Adult Functioning and Mortality

by

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Dissertation

Presented to the Faculty of the Graduate School of

The University of Texas at Austin

in Partial Fulfillment

of the Requirements

for the Degree of

Doctor of Philosophy

The University of Texas at Austin

August 2011

Acknowledgements

I am extremely grateful for the invaluable mentorship and training that I received from Mark Hayward and Bob Hummer consistently throughout the past five years. It has been an honor to learn from two absolutely brilliant, enthusiastic, and genuine individuals. Thank you for always being available for advice and answers, for your open-door policy, for remarkably prompt feedback on my research, for encouraging me to pursue my research interests, and for treating all of your students with upmost respect and as junior scholars. Bob, I came to The University of Texas at Austin because of you. As a prospective graduate student, I called you (the Department Head at that time) about the department and you discussed my decision with me for over an hour. I knew then and there that this was the right place for me. Mark, how can I thank you enough? Thank you for taking me under your wing, for (gently) challenging me and pushing me to think about issues in different ways, for your patience, and for always being available to discuss my work and professional decisions. I am amazed at how you find the time to provide stellar and constant mentorship to all of your graduate students while being the Director of the PRC. I will greatly miss our one-on-one meetings and our weekly working group meetings that I benefitted so much from. I trust that we will continue to collaborate and communicate often.

I am also thankful for the advice and encouragement I received from the three additional members of my dissertation committee. I especially want to thank Debra Umberson for the opportunity to work with you and learn from you. I will remember to "be bold" and I hope that we collaborate again and again. Thank you, Cynthia Osborne, for your quick and thorough feedback while I was writing my dissertation and for

reminding me to wear my policy hat when approaching my research. I am also thankful for the positive energy and support from Tetyana Pudrovska during my dissertation research and job search process.

I am very grateful to the Department of Sociology and the Population Research Center at The University of Texas at Austin for the academic training, mentorship, and financial support that I received during my graduate studies. I especially appreciate the financial support of the Eunice Kennedy Shriver National Institute of Child Health and Human Development pre-doctoral traineeship from the Population Research Center and the Mike Hogg Endowment Fellowship from the Graduate School. I am particularly thankful for the guidance of Mary de la Garza at the Population Research Center who helped me navigate each step of the doctoral process with her infinite expertise and charm.

I owe several amazing women whose constant encouragement and friendship supported me through my graduate studies and writing this dissertation. To Kari White, Karen Rascon, Dara Shifrer, and Andrea Henderson, thank you for being my rocks during the past five years, for giving endless pep talks, and keeping it all real.

Lastly, and most importantly, thank you mom and dad for encouraging me to pursue this, and so many other, dreams throughout my life. I have been blest with the most amazing parents. Thank you for taking a genuine interest in my research and listening to me talk about it incessantly for the last five years. None of this would have been possible without you. I love you.

Gender Differences in the Life Course Origins of Adult Functioning and Mortality

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The University of Texas at Austin, 2011

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A high degree of physical functioning is necessary for independently performing the numerous routine and valued tasks of daily life. Poor functioning not only hinders independent living, it can lower the quality of life, impede full social participation, and elevate the risk of death. However, not all adults are at equal risk of poor functioning: women experience worse functioning and live a greater number of years functionally impaired compared with men. Studies of this gap have focused on inequities in adult circumstances, such as socioeconomic status, but have generally fallen short of fully accounting for it. Recasting this research within a life-course, epidemiological framework points to the potential role of early-life circumstances. Early-life circumstances may impart a biological imprint, and they may also launch long-term trajectories of social circumstances, that could differentially shape functioning for men and women. Thus, this dissertation examines the life course origins of the gender gap in functioning and active life expectancy among older U.S. adults using two nationally-representative datasets: the National Survey of Midlife Development in the United States and the Health and Retirement Study. In sum, the findings reveal that: (a) a host of early-life circumstances,

such as parents' education levels, leave an indelible stamp on functional ability and active life expectancy for women and men, irrespective of adult circumstances, (b) while some early-life adversities, such as extreme poverty, were marginally more consequential for women's than men's functioning, they appear to be primarily more consequential for precipitating metabolic conditions such as diabetes and obesity rather than directly impacting functioning, (c) explanations of the gap must incorporate endogenous biological differences between men and women; explanations that focus exclusively on socially-structured inequities are insufficient, and (d) exposures to socioeconomic resources accumulate across the life course to shape functioning differently for men than women; particularly between white men, who enjoy better functioning with higher educational attainment irrespective of early-life socioeconomic exposures, and white women whose functioning gains plateau if they experienced early-life socioeconomic adversities. Overall, the results underscore the importance of a life course perspective in explicating gender disparities in functioning, longevity, and active life expectancy.

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Chapter 1: Introduction

MOTIVATION

Women can expect to live longer than men but experience worse health on many dimensions, particularly those reflecting physical functioning such as arthritis, functional limitations, and disability. To illustrate, in the United States in 2004, life expectancy at birth for women was 80.4 years compared with 75.2 years for men (Arias 2007). However, 24.4 percent of women and 19.1 percent of men 18 years and older reported some degree of physical impairment such as functional limitations, difficulty performing instrumental activities of daily living such as using a telephone, or difficulty performing basic activities of daily living such as bathing (Centers for Disease Control and Prevention 2009). The gender gap in functioning becomes even more pronounced with age (Gorman and Read 2006). For instance, among U.S. adults aged 51 to 61 years in the 1992 Health and Retirement Study, 23 percent of men and 33 percent of women reported difficulty with lower body functioning, while 20 percent of men and 44 percent of women reported difficulty with upper body functioning (Wray and Blaum 2001).

The preponderance of studies that have investigated the gender gap in adult functioning and mortality has focused on inequities in adult circumstances between men and women. Indeed, compared with men, women are more likely to experience economic hardship, be unemployed or employed in undesirable jobs, experience psychosocial stressors, be primarily responsible for raising children and caring for aging parents, and

be physically inactive (Ross and Bird 1994)—factors that, in turn, can deteriorate health. At the same time, men are more likely than women to engage in risky behaviors such as heavy cigarette smoking, reckless driving, and be employed in dangerous occupations—factors that, in turn, increase mortality risk. However, despite decades of scholarship aimed at unraveling the multifarious causes of gender disparities in functioning and mortality, studies that have focused on adult circumstances as potential explanations have largely fallen short of fully accounting for the disparities.

Growing alongside the long tradition of research on the gender gap in health is a separate and relatively more recent area of research on the early-life origins of adult health. This latter line of inquiry has been generating mounting evidence that early-life circumstances such as prenatal nutrition, childhood health, pathogen exposures, socioeconomic environment, and family disruption leave an indelible imprint on a host of later-life health conditions (e.g., diabetes, obesity, cardiovascular disease, and functional impairment) and mortality risk, independent of adult circumstances. Thus, adult health and longevity does not appear to simply reflect proximal circumstances: rather, recent evidence from the United States and many European countries convincingly documents that adult health and longevity reflect the accumulation of physical (e.g., nutrition, infectious disease) and social (e.g., education, social ties) exposures that occur during gestation, childhood, adolescence, and adulthood (Barker 1997; Kuh and Ben-Shlomo 2004; Montez and Hayward 2011). Is it conceivable, then, that early-life conditions contribute to the substantial gender gap in adult functioning and mortality risk? In other words, should scholars search further upstream in the life course for an explanation?

One of the major weaknesses in prior research on the early-life origins of later-life health and mortality is that most studies have been based on data that consists exclusively of men, or data that consists of both men and women but analyzed with a simple statistical control for gender. Thus, we know little about whether early-life exposures have similar implications for men's and women's later-life health and mortality. A differential effect is theoretically plausible. Early-life conditions may "interact" with sex differences in growth (e.g., bone development), maintenance (e.g., immune systems), and reproductive function that reflect sex-specific, life history strategies during a critical and highly plastic developmental window (Decaro, Decaro, and Worthman 2010). Despite sound theoretical reasons to suspect a differential effect between men and women, a dearth of studies has formally investigated the possibility. Based on a small handful of studies that actually tested for a differential effect, it seems that some early-life adversities such as poverty may, in fact, have more pronounced physiological and structural consequences for women—for example, on their risks of diabetes (Maty et al. 2008), obesity (Heraclides, Witte, and Brunner 2008; Khlat, Jusot, and Ville 2009; Ravelli et al. 1999), cardiovascular disease (Hamil-Luker and O'Rand 2007), metabolic syndrome (Langenberg et al. 2006; Lehman et al. 2005), and osteoarthritis (Kin et al. 2007)—although these studies are few in number and there is considerable ambiguity surrounding their findings.

This dissertation will integrate these two lines of research (gender differences in adult health and mortality; the early-life origins of adult health and mortality) into a life course epidemiological framework to investigate whether the substantial gap in physical

functioning, mortality risks, and active life expectancy between adult men and women is anchored in early life. In other words, are adult women more likely than adult men to have experienced early-life adversities that have long-term consequences for their health? Are women more "reactive" than men to adversities experienced in early life such that the long-term, health consequences are more pronounced among women? If women are indeed more reactive than men to early-life adversities, what mechanisms are involved? For instance, it might be the case that early-life adversities, such as poverty, are simply more likely to impede women's than men's upward social mobility, such that the explanation points to socially-structured constraints and opportunities. Alternatively, it might be the case that early-life adversities impart a more extreme biological stamp on women's physiological systems and structural constitutions than they do for men, such that the explanation points to inherent biological differences. These are the overarching questions motivating the research within this dissertation.

The life course framework implemented here is imperative for locating the origins of the gender gap in adult functioning and mortality, revealing the intervening processes that link distal and proximal exposures with these outcomes, and designing appropriately targeted, public health interventions. Among the many advantages of integrating early-life conditions into research on adult health disparities, two are particularly important. First, to the extent that early-life conditions shape baseline functioning and mortality risk, they are crucial for explicating variations in mid-life health and the nature of health declines thereafter. Indeed, the link between socioeconomic status and adult health, as well as the link between social relationships and adult health, is stronger when using

measures that combine childhood and adult experiences compared with single point in time measures (Seeman et al. 2002; Singer and Ryff 1999). Second, integrating circumstances from the entire life course can reveal the critical periods for specific social and physical exposures on specific health conditions (e.g., poor hygiene in the childhood home and the risk of death from stomach cancer in later life), and therefore guide policy initiatives to have the most efficacious impact on population health and reduce health disparities.

The research presented here focuses on three health-related outcomes for which men and women exhibit dramatic differences: physical functioning, all-cause mortality risk, and active life expectancy. These outcomes were also selected in part because they are substantively meaningful. For instance, a high degree of physical functioning is necessary for independently performing the numerous routine and valued tasks of daily life. Poor functioning not only hinders independent living, it can lower quality of life, impede full social participation, and elevate the risk of death (Melzer, Lan, and Guralnik 2003). In addition, poor functioning can be financially costly for individuals, their families, and governments. Over 300 billion dollars have been spent annually in the United States since 1994 on disability-related costs for medical care and lost productivity (U.S. Department of Health and Human Services 2005). In addition, mortality is also a particularly relevant and "ultimate" health outcome. Lastly, active life expectancy reflects the intersection of physical functioning and mortality risk. It quantifies the number of years an individual can expect to live free from functional impairment and is thus an exceptional indicator of both length and quality of life.

RESEARCH QUESTIONS

This dissertation addresses many of the gaps outlined above throughout three separate but related chapters. The analysis presented in Chapter Two examines the extent to which early-life socioeconomic conditions and family relationships predict functional limitations among adults in midlife (45 to 74 years of age), whether these early-life conditions differentially predict functional limitations for women compared with men, and which of the major social and biological mechanisms most strongly link these early-life conditions to functioning. The analysis is unique in that it: (a) integrates a broad set of theoretically important early-life conditions (e.g., parents' education, poverty experience, relationship quality with mother, childhood health) in order to identify the strongest predictors of functional limitations in adulthood, and (b) aims to identify which of the commonly hypothesized, social and biological mechanisms most clearly link these early-life conditions with functional limitations in ways that potentially differ between women and men. The chapter uses the first wave of data from the Midlife Development in the United States Survey, which is a cross-sectional, nationally-representative survey of men and women 25 to 74 years of age conducted in 1994-1995. The research questions addressed in chapter two are listed below.

1. To what extent are early-life socioeconomic and family exposures associated with functional limitations among middle-aged U.S. adults, and how do these associations differ for men and women?
2. Which social pathway and biological imprint processes link early-life exposures with functional limitations, and how do the links differ for men and women?

Chapter Three focuses on a single, albeit powerful, early-life condition—socioeconomic resources as measured by parents' education levels—and examines the extent to which it predicts transitions between various states of functional health (no functional impairment, functional limitations, difficulty with instrumental activities of daily living, and difficulty with basic activities of daily living), between functional health states and death, and active life expectancy, net of adult socioeconomic conditions; and whether the extent differs between women and men. Chapter Three is unique in that it: (a) is the first known study to simultaneously examine whether and how early-life socioeconomic conditions predict functioning across multiple functioning states represented in the Disablement Process (Verbrugge and Jette 1994) as prior studies have evaluated only a single stage of the process, and (b) examines how early-life socioeconomic conditions predict the intersection of functioning and mortality risk, a concept called active life expectancy (prior studies have only evaluated these two outcomes separately). It uses data from the 1998 through 2008 Health and Retirement Study, which is a longitudinal panel study of U.S. adults 50 years of age and older, and their spouses. The research questions addressed in Chapter Three are listed below.

1. To what extent are transitions between functioning states within the disablement process related to parents' education and how do the associations differ between men and women?
2. To what extent is active life expectancy related to parents' education and how does the association differ between men and women?

3. Does adjusting for parents' education help explain the gender gap in functioning across the disablement process and in active life expectancy?
4. To what extent is the proportion of life spent functionally impaired related to parents' education and how does this differ for men and women? Do subgroups with highly-educated parents exhibit a compression of functional impairment within the life span?
5. To what extent does own educational attainment mediate the association between parents' education and the above health outcomes?

Consistent with Chapter Three, Chapter Four focuses on early-life socioeconomic resources (as measured by parents' education levels) as the sole indicator of early-life conditions. It is unique in that it: (a) evaluates how exposure to early-life socioeconomic conditions and adult socioeconomic conditions accumulate to predict functional limitations and all-cause mortality risk, and (b) assesses whether the accumulation differs for men compared with women, and for blacks compared with whites. In other words, do socioeconomic resources combine additively such that the health-related benefits of adult socioeconomic resources are in addition to, and irrespective of, early-life socioeconomic resources, or do they combine interactively such that the health-related benefits of adult socioeconomic resources are contingent on one's early-life socioeconomic resources; and how does nature of the accumulation differ for men compared with women, and for whites compared with blacks? It uses data from the 1992 through 2008 Health and Retirement Study, which is a longitudinal panel study of U.S. adults 50 years of age and

older, and their spouses. The research questions addressed in Chapter Four are listed below.

1. To what extent are early-life and adulthood socioeconomic resources associated with functional limitations and all-cause mortality risk in later life?
2. Do early-life and adult socioeconomic resources accumulate additively or interactively on later-life functioning and all-cause mortality risk? If they accumulate interactively, does the data support a Synergistic or Catch-Up hypothesis? Does the data further support biological imprint and/or social pathway hypotheses in explaining the link between early socioeconomic resources and these two outcomes?

OVERVIEW OF DATA SOURCES

The analyses utilize two well-established data sources: the National Survey of Midlife Development in the United States (MIDUS) and the Health and Retirement Study (HRS). Each source has strengths and weaknesses for addressing the proposed research questions. The data sources are described in detail below.

MIDUS is a longitudinal panel study of U.S. adults aimed at examining the role of behavioral, psychological, and social factors in generating physical and mental health outcomes across the adult life course. To date, MIDUS contains two waves of data. The first wave was conducted in 1994-95 and contains 7,108 adults 25 to 74 years of age. The 3,032 respondents for the main sample were recruited from a representative random-digit-dial phone sample of non-institutionalized, English-speaking adults 25 to 74 years

of age living in the contiguous United States. The remaining 4,076 respondents were recruited from sibling-, twin-, and city- oversamples. The second wave was conducted in 2004-2006 and contains 4,963 of the adults from the 7,108 interviewed in wave 1. In this dissertation, I rely exclusively on wave 1 in order to maximize the sample size. The main strengths of MIDUS for the current analysis include the exceptionally rich information on retrospectively-recalled childhood circumstances—for example, perceived quality of relationships with one's parents in childhood, information on mother's and father's education and occupation, childhood health, family structure, residential mobility, and discipline and affection from parents. However, MIDUS is not a good source for mortality analyses given the small number of deaths in the sample, or for describing long-term health trajectories given that it is currently limited to two waves of data collection.

The HRS is a longitudinal survey of older U.S. adults aimed at examining the changing health, family, and economic situations of adults as they make the transition to retirement, and then again between post-retirement and the end of life. The original HRS consisted of three waves of data collection in 1992, 1994, and 1996 among adults born between 1931 and 1941. In 1998, the HRS was integrated with a companion study—the Assets and Health Dynamic among the Oldest Old (AHEAD)—that was conducted in 1993 and 1995 among adults born between 1890 and 1923. At that time, the sampling frame was expanded to include two additional cohorts, the Children of the Depression Era (CODA) born 1924 to 1930 and War Babies (WB) born 1942 to 1947, and the survey instrument was expanded to include retrospectively-reported information about childhood circumstances. While information on mother's and father's education was collected in all

waves, all other early-life information—such as childhood health and poverty experience—began in 1998. Thus, analyses that require detailed information on childhood conditions must begin in 1998. The main strengths of the HRS for the current analysis include its large sample size, the number of survey waves, and a large number of deaths for analyses concerned with mortality risk and active life expectancy. On the other hand, the age-range of the HRS is limited for certain life course analyses, and information on childhood family structure and relationships is unavailable.

Chapter 2: Gender Differences in the Early-Life Origins of Midlife Functional Health

CHAPTER 2 ABSTRACT

Research on women's higher prevalence of functional limitations compared with men has focused on the role of adult circumstances, but has largely fallen short of accounting for the disparity. Because mounting evidence finds that early-life conditions exert an enduring influence on adult health, I hypothesize that the disparity in functioning may originate in childhood. This chapter uses data from the National Survey of Midlife Development in the United States to identify the early-life origins of functional limitations among adults 45 to 74 years of age, and to evaluate intervening social and biological processes. Results indicate that functional limitations, and their gender gap, are partially anchored in early life. In particular, early-life poverty predicted poorer functioning among men and women by depressing educational attainment and elevating negative affect; and through metabolic and cardiovascular conditions among women, net of adult confounders. The findings underscore the importance of a life course approach in explicating gender disparities in functioning.

Women are more functionally impaired than men. For instance, among adults aged 51 to 61 years in the 1992 Health and Retirement Study, 23 percent of men and 33 percent of women reported difficulty with lower body functioning, while 20 percent of men and 44 percent of women reported difficulty with upper body functioning (Wray and Blaum 2001). Studies of this gender gap have traditionally focused on adult circumstances such as socioeconomic status and precipitating chronic conditions, but have generally fallen short of explaining women's higher prevalence. Mounting evidence that early-life circumstances, such as childhood nutrition and parental divorce, leave an indelible imprint on adult health—along with possible gender differences in exposure and reaction to those conditions—suggests that early-life circumstances may anchor the gap in functioning in important ways. Whether, how, and why early-life conditions anchor the gap is poorly understood. These are the questions motivating this chapter.

THEORY AND EVIDENCE

A high degree of physical functioning is necessary for independently performing the numerous routine and valued tasks of daily life—tasks that many Americans take for granted such as walking to the mailbox or climbing a flight of stairs. Poor functioning not only hinders independent living, it can lower quality of life, impede full social participation, and elevate the risk of death (Melzer et al. 2003). In addition, poor functioning can be financially costly for individuals, their families, and governments. Over 300 billion dollars have been spent annually in the United States since 1994 on disability-related costs for medical care and lost productivity (U.S. Department of Health

and Human Services 2005). Thus, identifying the social, behavioral, and biological risk factors for poor physical functioning could result in more effective public health interventions, improved population health, and substantial cost savings.

In the United States in 2005, 17.3 percent of civilian, noninstitutionalized adults aged 18 and older reported some degree of functional limitation (Centers for Disease Control and Prevention 2009). However, not all Americans are at equal risk of functional limitations: older adults, women, racial/ethnic minorities, the unmarried, and persons of low socioeconomic status (SES) experience disproportionate risks (Freedman and Martin 1998). During middle and later life, women especially bear a disproportionate risk. Despite a large literature examining women's greater risk (whether measured by functional limitations or disability), there is currently no widely accepted explanation for the mechanisms underlying their functioning disadvantage.

Studies of the gender gap in functioning have historically focused on gender differences in the likelihood and consequences of precipitating chronic conditions in adulthood (e.g., Gorman and Read 2006; Murtagh and Hubert 2004; Oman, Reed, and Ferrara 1999; Strawbridge et al. 1993; Wray and Blaum 2001). These studies generally find that, among the major precipitating conditions for functional limitations, three of them are indeed disproportionately experienced by women: musculoskeletal conditions, obesity, and depressive symptoms (Murtagh and Hubert 2004; Wray and Blaum 2001). Further, some conditions appear more consequential for functioning among women than men: musculoskeletal conditions (Murtagh and Hubert 2004; Verbrugge and Patrick 1995), obesity (Wray and Blaum 2001), diabetes (Verbrugge and Patrick 1995) and

possibly high blood pressure (Verbrugge and Patrick 1995). However, with few exceptions (Murtagh and Hubert 2004), accounting for these chronic conditions, and social and behavioral risk factors, does not fully explain the gender gap in adult functioning (Alvarado, Guerra, and Zunzunegui 2007; Gorman and Read 2006; Wray and Blaum 2001).

Surprisingly few studies of the gender gap in functioning have been based on a life course perspective. This theoretical and empirical void is curious because functional limitations and their precipitating chronic diseases usually develop over protracted periods of time, and may reflect the accumulation of exposures, events, and behaviors across the life span. In reality, gender differences in functioning may reflect a complex interplay of sex differences in biological vulnerabilities (e.g., bone mass and reproductive function) across the life span with gender differences in social experiences (e.g., gender norms for body weight, occupations) across the life course. A life course perspective is imperative for locating the origins of the gender gap, revealing the intervening processes that link distal and proximal exposures with functioning, and designing appropriately targeted, public health interventions. In other words, simply implicating gender disparities in arthritis prevalence as a major contributor to the gap in functioning does not indicate whether the causal processes originate in childhood, adulthood, or both.

The need for a life course perspective for unraveling the origins of functional limitations—and their gender differences—is underscored by emerging evidence that early-life exposures have an enduring influence on functional limitations (Alvarado et al. 2007; Guralnik et al. 2006; Haas 2008; Haas 2007; Luo and Waite 2005; Turrell et al.

2007) and their precipitating chronic diseases (Blackwell, Hayward, and Crimmins 2001; Hamil-Luker and O'Rand 2007; Kuh and Ben-Shlomo 2004). Conceptually, the influence of early-life exposures may operate through "direct" and "indirect" processes. They may directly shape functional health through biological imprint processes. For example, prenatal nutrition may permanently alter the structure and function of organs and tissues—particularly those associated with metabolic and cardiovascular systems (Barker 1997). Early-life exposures may also indirectly shape functional health through social pathway processes. For instance, early-life SES may initiate long-term trajectories of social, psychological, and behavioral (dis)advantages, and the proximate adult (dis)advantages may then shape functional health (Lundberg 1993; Palloni et al. 2009). Supporting both processes, studies in the U.S. (Haas 2008; Haas 2007; Luo and Waite 2005; Turrell et al. 2007), Britain (Guralnik et al. 2006), and Latin America (Alvarado et al. 2007) find that early-life SES predicts functional health, and that intervening mechanisms only partly mediate the associations.

Given the substantial gender gap in functioning, however, it is surprising how little is known about how early life shapes functioning for women compared with men. The seminal research on the early-life origins of later-life health was largely based on single sex studies (usually of men), or studies that combined men and women and statistically controlled for gender. Only a few studies have tested for gender differences in the strength of the associations between early-life factors and later-life functioning, and the results are mixed. Among studies that find significant gender differences in the strength of the associations, they generally report that adverse early-life exposures are

more consequential for women's than men's functional health. Specifically, low SES (cf. Luo and Waite 2005; Turrell et al. 2007), poor health (Luo and Waite 2005), and inadequate nutrition (Alvarado et al. 2007) in early life may be more consequential for women. If early-life adversities are indeed more consequential for women than men, then early-life exposures may partially anchor the gender gap in functioning.

We also know very little about which social pathway and biological imprint processes link early-life exposures with adult functioning, and whether these processes differ for men and women. In fact, the three studies mentioned above did not systematically examine intervening processes. Thus, many questions remain unanswered. Are early-life exposures more likely to impart a physiological scar on women than men, which then compromises functioning? Are early-life exposures more likely to impede socioeconomic attainment, psychosocial well-being, and health behaviors among women than men, which then compromise functioning? Below, I hypothesize theoretically important social pathway and biological imprint processes that may link early-life exposures with functioning in ways that differ for men and women.

Social Pathway Processes

One major social pathway, socioeconomic achievement, may indirectly influence functional limitations by placing people into an adult SES that mirrors their childhood SES; and adult SES may then shape functional limitations. Indeed, adult SES predicts many of the health behaviors and chronic diseases that are precursors for functional limitations. Because access to higher education and lucrative occupations has historically

been greater for men than women, some scholars speculate that adverse early-life SES may be more consequential for women's functional health because women have been less able to escape early-life economic adversity through their own upward mobility (Hamil-Luker and O'Rand 2007).

Early-life exposures may indirectly shape functioning through psychosocial pathways. For instance, Shaw and colleagues (2004) found that a lack of emotional support from parents in early life predicted chronic conditions and depressive symptoms (both are precursors for functional limitations) in adulthood, and that the association was largely explained by adult psychosocial characteristics, including a low sense of control, low self-esteem, and poor quality social ties. Other scholars similarly assert that a harsh childhood family environment shapes adult health through negative emotionality and low social competence in adulthood (Taylor et al. 2004). Moreover, the links between early-life exposures and psychosocial well-being may differ for men and women. A study of Finnish adults found that among 21 types of retrospectively recalled childhood experiences, nine were associated with adult depression and two (neurotic symptoms, poor mother-child relationship) were stronger predictors of depression among women than men (Veijola et al. 1998). Others have found that parental divorce has more negative psychosocial consequences for boys than girls (Amato 2005).

Early life can also indirectly shape functioning through a behavioral pathway. Adults raised in environments characterized by economic adversity, harsh and unsupportive family interactions, family dysfunction, or abuse are more likely to smoke, abuse alcohol and illicit drugs, engage in sexually promiscuous behavior, be physically

inactive, and be obese as adults (Felitti et al. 1998; Lynch, Kaplan, and Salonen 1997; Repetti, Taylor, and Seeman 2002). These negative health behaviors, in turn, compromise functional health.

Biological Imprint Processes

Early-life exposures may directly shape functional limitations through a permanent imprint on the musculoskeletal system (Gale et al. 2001). For instance, bone mass in later life is a function of peak bone mass obtained during skeletal growth and the subsequent rate of bone loss (Javaid and Cooper 2002). The role of early-life exposures is clear here, as peak bone mass is influenced by sex, heredity, and early environmental factors such as nutrition and exercise (Javaid and Cooper 2002). Indeed, early-life nutrition predicts bone structure and osteoarthritis risk in later life, net of adult risk factors for bone loss (Javaid and Cooper 2002; Kin et al. 2007; Woo, Leung, and Wong 2010), and the association may be stronger for women than men (Kin et al. 2007). It may be stronger for women because, on average, women start life with lower bone mass than men, controlling for weight (Rupich et al. 1996), and because gender-specific norms for body weight and physical activity in childhood and adolescence may exacerbate the sex difference. In addition, poor postnatal nutrition and adverse childhood SES have been linked with an earlier age of menopause, net of adult confounders (Hardy and Kuh 2002, 2005): early menopause increases the years that women are exposed to the risk of low bone density and osteoporosis (see review in Shuster et al. 2010).

Early-life exposures may directly shape metabolic function. David Barker and colleagues proposed the "thrifty phenotype" hypothesis that prenatal nutrition permanently alters metabolic and cardiovascular systems, and predisposes adults to such diseases (Barker 1997; Hales and Barker 1992). Moreover, early-life adversities may be more consequential for metabolic conditions among women than men. Ravelli and colleagues (1999) found that women prenatally exposed to the Dutch famine were more likely to be obese at age 50 than women who were not exposed—irrespective of childhood SES, adult SES, and health behaviors—while the association did not hold for men. Also, birth weight (a marker of prenatal nutrition) is inversely related to later risk of gestational diabetes (Innes et al. 2002). In addition to early nutrition, childhood SES may be a stronger predictor of adult obesity (Heraclides et al. 2008; Khlat et al. 2009), diabetes (Maty et al. 2008), and metabolic syndrome (Langenberg et al. 2006; Lehman et al. 2005) among women than men, net of adult confounders. Many of these studies implicate a sex-specific biological imprint on metabolic function. Also noteworthy, experimental studies with rats suggest that male-female differences in the effect of early-life nutrition on adult rat diabetes risk reflects sex differences in metabolic function (Dahri et al. 1995; Moura, Pereira, and Mandarim-de-Lacerda 2003). However, behaviors may also play a role. Low SES predicts inactivity among low SES girls somewhat more than boys (Lee, Harris, and Gordon-Larsen 2009). Other scholars point out differences in the symbolic meaning of body size among low SES adults (Khlat et al. 2009), and higher parity among low-SES women.

A third key physiological mechanism is cardiovascular diseases (CVD). The inverse association between early-life SES and adult CVD is well-established, and it is often only partly mediated by adult risk factors (Galobardes, Davey Smith, and Lynch 2006). Indeed, several early-life exposures that co-occur with low SES—inadequate nutrition (Barker 1997), infectious disease burden (Cohen et al. 2004; Dowd, Zajacova, and Aiello 2009), and psychosocial stress (Repetti et al. 2002; Taylor et al. 2004; Taylor, Repetti, and Seeman 1997)—may permanently alter the cardiovascular system. There is some indication that the strength of the associations differs for men and women, although neither gender seems clearly advantaged. For instance, childhood SES may be more strongly linked with CVD among women than men (Galobardes et al. 2006; Hamil-Luker and O'Rand 2007); while harsh early-life family environments may be linked to high cardiovascular reactivity among men but not women (Taylor et al. 2004).

Taken together, prior research suggests that: (a) the origins of adult functional health may be located in very early life, (b) adult functional health may reflect the accumulation of exposures throughout the life course, (c) the multifarious links between early-life exposures and adult functioning may reflect biological imprint and social pathway processes, and (d) the strength of the associations between early-life exposures and adult functioning —as well as the intervening processes—may differ for men and women. This chapter examines these hypotheses and addresses the following two research questions:

1. To what extent are early-life socioeconomic and family exposures associated with functional limitations among middle-aged U.S. adults, and how do these associations differ for men and women?
2. Which of the hypothesized social pathway and biological imprint processes link early-life exposures with functional limitations, and how do the links differ for men and women?

DATA AND METHODS

Data

The National Survey of Midlife Development in the United States (MIDUS) is a panel study of adults aimed at examining the behavioral, psychological, and social causes of adult health. Here, I draw on the 1994 baseline wave of MIDUS because it contains a larger sample than the second wave, and because cross-sectional data can effectively address the current research questions. The main sample contains 3,032 adults and is nationally representative of non-institutionalized, English-speaking adults 25 to 74 years of age. MIDUS is well-suited for the current study because of its rich data on (retrospectively recalled) early-life socioeconomic and family environments.

Because the focus of this chapter is on midlife and beyond, the analytic sample retains the 1,668 adults aged 45 to 74 years. The sample then excluded students, foreign-born adults, and adults who were missing data on gender, race, or functional limitations. Students were excluded because education is a key variable of interest. Foreign-born adults were excluded to minimize heterogeneity due to the possibility of education

obtained abroad and potentially fundamental differences in early-life exposures. The final analytic sample contains 1,527 adults.

Functional Limitations

Functional limitations were measured as the average degree of self-reported difficulty in performing four actions (Hubert, Bloch, and Fries 1993). Self-reports are a fairly accurate reflection of actual ability for men and women (Merrill et al. 1997). Respondents were asked, "How much does your health limit you in: climbing several flights of stairs; walking more than a mile; bending, kneeling, or stooping; walking several blocks." Responses included: a lot (3), some (2), a little (1), not at all (0). The functional limitations measure is the average of the four responses. A full 56.8 percent of the analytic sample reported some degree of functional limitations.

Early-Life Exposures

This study includes several theoretically important early-life exposures. In preliminary analyses, I determined how to specify certain exposures by testing for differences in functional limitations between measured categories. The first group of exposures reflects early-life SES. Having a low-educated father and low-educated mother are two binary indicators (1=less than high school diploma or education unknown; 0= high school diploma or more). Having a father with a low-status occupation and a mother with a low-status occupation are also binary indicators (1= blue collar, never or rarely

employed, or occupation unknown; 0=white collar).[1] Perceived income is the respondent's perception about how much worse or better off their childhood family was than the average family at that time, on a 7-point scale. Poverty indicates whether there was ever a period of six months or more that their family received welfare. The next group reflects psychosocial resources. Intact indicates whether they lived with both biological parents until at least 16 years of age. Respondents rated their relationship with their mother during childhood on a 5-point scale with higher scores reflecting better relationships. A similar question regarding fathers showed no association with functioning. The third block includes an indicator of fair or poor childhood health at 16 years.

Adult Exposures

This study includes well-established adult exposures that predict functional health (Stuck et al. 1999) and test the hypothesized social and biological processes. The first group reflects adult SES, measured by educational attainment. Education is measured with three dummy variables: less than high school, high school diploma or some college (omitted reference), or bachelor's degree and higher.[2] The next block reflects psychosocial resources. Marital status is a binary indicator (1=married or cohabiting).

[1]White collar includes executive/administrative/managerial; professional specialty; technician; sales; administrative/clerical support. Blue collar includes service; farming/forestry/fishing; production/craft/repair; operator/laborer/military.

[2]Preliminary analyses included household income. The final analysis did not include income because income did not mediate, to any degree, the association between early life exposures and functional limitations, nor did it mediate the association between educational attainment and functional limitations, and many respondents did not report income.

Negative affect ranges from one to five and is the mean response to six questions (α=0.86): How much of the time during the last 30 days did you feel so sad nothing could cheer you up; nervous; restless or fidgety; hopeless; that everything was an effort; worthless. Mastery ranges from one to seven and is the mean response to four questions (α=0.69): How strongly do you agree or disagree with the following statements, I can do just about anything I really set my mind to; when I really want to do something, I usually find a way to succeed at it; whether or not I am able to get what I want is in my own hands; what happens to me in the future mostly depends on me. The third block captures behaviors. For women, parity ranges from zero to five or more. Pack-years are the ln(packs of cigarettes smoked per day times the number of years as a smoker). Obese indicates a BMI of 30 or higher. Because there is some debate about the extent to which obesity reflects behaviors or biological set-points, and because the analysis finds early-life poverty predicts obesity for women, ancillary analyses control for two global health attitudes (how much thought and effort do you put into your health these days; to what extent do you work hard at trying to stay healthy), to glean some insights into the debate. The attitudes are not included in the main analysis because of missing data. The last block includes indicators of key chronic conditions: arthritis, hypertension, diabetes, heart trouble, and major depressive episode.

Analytic Strategy

The analysis first provides the distributions of early-life and adult exposures, and the associations between each exposure and functional limitations. The analysis then

builds gender-stratified OLS models that sequentially include groups of exposures. The models first identify which early-life exposures are independently associated with functional health by sequentially including early-life exposures in their presumed temporal order (e.g., father's education is included before father's occupation) and retaining exposures significant at $p<0.10$. Next, the models aim to identify social and biological processes that explain the associations by then sequentially including adult exposures and retaining exposures significant at $p<0.10$. Empirical support for biological imprint and social pathway processes are described below. To be clear, the analysis cannot unequivocally adjudicate between social and biological processes. Rather, it aims to identify plausible processes for further investigation. All analyses were conducted with SAS Version 9.2, and were weighted and adjusted for race (white, nonwhite) and age. All non-binary variables are centered at their gender-specific mean.

Empirical Support for Social Pathway Processes

- Socioeconomic: The associations between early-life exposures and functional limitations attenuate when adult SES is included.
- Psychosocial: The associations attenuate when adult psychosocial factors are included.
- Behavioral: The associations attenuate when adult health behaviors are included.

Empirical Support for Biological Imprint Processes

The associations between early-life exposures and functional limitations do not attenuate when all adult exposures are included; or they attenuate when a chronic condition is included net of key behavioral, socioeconomic, and psychosocial risk factors for that condition.

RESULTS

Among women, the average age was 58.2 years, and 11.6 percent were nonwhite. Among men, the average age was 57.3 years, and 12.1 percent were nonwhite. For each exposure, Table 2.1 shows its distribution and its association with functional limitations (FL). Gender differences in the distributions or in the associations indicate that the origins of the gender gap are at least partially anchored in early life. Compared with men, women reported poorer relationships with their mothers during early life and they were more likely to report a low-educated mother.[3] The associations between four early-life exposures (father's education, mother's occupation, mother-child relationship, and childhood health) and FL differed between men and women such that the exposures were more consequential for women's functioning. For instance, while having had a low-educated mother was associated with a 0.09 ($p<0.10$) increase in FL similarly for women

[3]The low education group combines "less than high school" and "don't know" responses for mother's education because these respondents reported similar functional limitation scores. Disaggregating the two responses confirmed that women were more likely than men to report their mother had less than a high school education (49.8% versus 42.2%), while they were not more likely to report that they did not know their mother's education (10.7% versus 9.7%). Differential recall may play a role. Mortality selection is less likely because the disparity also existed among the 25-44 year adults in MIDUS. Also note that women were more likely than men to report their father belonged to the combined "less than high school or don't know" group, yet this difference was not significant among the disaggregated responses (e.g., 48.4% of women and 46.0% of men reported their father had less than a high school education). These patterns highlight the need for additional validation studies of retrospective recall of early life conditions.

and men, having had a low-educated father was associated with a 0.07 ($p>0.10$) increase for men and a 0.20 ($p<0.01$) increase for women. With few exceptions, women were also more likely than men to report adult exposures related to poor function, and many adult exposures were more consequential for women than men.

Early-Life Origins of Women's Functional Health

Table 2.2 contains coefficients from OLS models predicting FL based on the 704 women with complete data on all exposures.[4] In sum, the table reveals that three early-life exposures—father's education, mother-child relationship, and poverty—were strong predictors of FL. Father's education operated mainly through women's educational attainment, reflecting a socioeconomic pathway. The mother-child relationship operated through women's negative affect, which may reflect a psychosocial pathway. Early-life poverty operated partly through educational attainment and negative affect; and partly through obesity, diabetes, and heart trouble somewhat, which may reflect biological imprint processes. Figure 2.1 illustrates these key mechanisms. The next section describes Table 2.2 in detail.

According to model 1 in Table 2.2, the average FL score among women was 0.89. Models 2-6 examine which early-life SES indicators predicted FL, net of each other. To start, father's education was a significant predictor (model 2); and while parental occupations slightly mediated its association (model 3), they were not significant, net of

[4]Of the 1,527 male and female respondents, 148 were missing data on at least one exposure, with almost one-half (N=71) missing data on obesity. Compared with the 1,379 respondents with complete data, these 148 respondents had worse functional health and they were more likely to be older, female, nonwhite, and low-educated.

father's education. Models 4-6 reveal that early-life poverty was a better predictor than perceived parental income, and that father's education operated partly through early-life financial well-being. Next, among the two early-life psychosocial exposures tested in models 7-8, only the mother-child relationship predicted FL. Lastly, net of these exposures, early-life health no longer significantly predict FL (model 9).

Models 10-18 incorporate adult exposures to help identify social and biological processes that link the three early-life exposures—father's education, mother-child relationship, and poverty—with FL. The role of father's education on FL operated through adult educational attainment, demonstrated by the attenuation of father's education from 0.15 ($p<0.10$) in model 8 to 0.04 ($p>0.10$) in model 10. The mother-child relationship operated through negative affect, evidenced by its attenuation from -0.07 ($p<0.05$) in model 10 to -0.01 ($p>0.10$) in model 11. Early-life poverty operated through several processes. From a coefficient of 0.48 ($p<0.01$) in model 8, it was slightly attenuated to 0.42 ($p<0.01$) by educational attainment in model 10, to then 0.32 ($p<0.05$) by negative affect in model 11, to then 0.26 ($p<0.05$) by obesity in model 13. Further adjusting for heart trouble alone marginally reduced the poverty coefficient to 0.24 ($p<0.10$), while adjusting for diabetes alone reduced it to 0.20 ($p>0.10$). Obesity, heart trouble, and diabetes may reflect biological imprints because the associations were net of key social and behavioral risk factors for those conditions.

Early-Life Origins of Men's Functional Health

Table 2.3 repeats the previous analysis but includes the 675 men with complete data. In sum, the table reveals that just one early-life exposure, poverty, was particularly important, and that it operated through educational attainment and negative affect in adulthood, which may reflect socioeconomic and psychosocial pathways, respectively. Figure 2.2 illustrates these key mechanisms. A detailed description follows.

According to model 1 in Table 2.3, the average FL score among men was 0.61. For men, neither parents' education nor occupation predicted FL (models 2-3). The only significant early-life SES indicator was poverty. Note the poverty coefficient (0.25, $p<0.05$, in model 5) is roughly one-half the size it was for women (0.50, $p<0.01$, in model 6 of Table 2). Net of poverty, early-life psychosocial resources (models 6-7) and health (model 8) did not predict FL. Next, models 9-16 find that early-life poverty was mediated slightly by educational attainment from 0.25 ($p<0.05$) in model 5 to 0.22 ($p<0.10$) in model 9, and then to 0.11 ($p>0.10$) by negative affect in model 10.

DISCUSSION

The aim of this study was to identify the early-life origins of adult functional limitations—and their gender differences—by employing a biosocial, life course perspective. The study found that among U.S. adults aged 45 to 74 years in 1995: (a) several early-life conditions predicted adult functioning, and some early-life conditions were stronger predictors among women than men, (b) when examining early-life conditions concomitantly, only poverty was associated with functioning among men; while father's education, poverty, and the mother-child relationship were important

among women, (c) early-life conditions shaped adult functioning partly through socioeconomic (educational attainment) and psychosocial (negative affect) pathways for men and women; and partly through women's metabolic and cardiovascular systems. Thus, early-life conditions indeed appear to at least partially anchor the gender gap in later-life functional limitations.

This study underscores the importance of investigating the life course origins of adult health with explicit attention to sex and gender variation in these processes. While many scholars and funding agencies have advocated greater attention to sex and gender differences in the etiology of disease, disability, and death, the seminal literature on the early-life origins of these outcomes has paid relatively little attention to potential sex and gender variations in the processes. A handful of studies has recently begun investigating these processes separately for women and men (e.g., Hamil-Luker and O'Rand 2007; Khlat et al. 2009; O'Rand, Hamil-Luker, and Elman 2009). The current study corroborates emerging evidence that early-life conditions shape adult men's and women's health in ways that appear more varied, consequential, and enduring for women. The current study also greatly extends prior research by developing and testing a biosocial, life course framework for identifying the mechanisms through which early-life conditions shape later-life health in sex/gender-specific ways.

Among women, several early-life conditions—particularly father's education, mother-child relationship, and poverty—were important predictors of functioning, and they were more important for women's than men's functioning. Women raised by low-educated fathers were more functionally impaired than other women because they

attained low levels of education as adults (a socioeconomic pathway). Moreover, having had a low-educated father was more consequential for women's than men's functioning. The gender differential existed because own educational attainment was more consequential for women's than men's functioning—not because these women were less likely than men to be upwardly mobile (Spearman's correlation based on four ordinal levels of father's education and four ordinal levels of adult's education was 0.414 for men and 0.416 for women). In addition, a poor mother-child relationship impaired women's functioning by elevating negative affect, reflecting a psychosocial pathway. This finding corroborates a study of Finnish adults, which found that a poor mother-child relationship was a stronger predictor of depression among women than men in adulthood (Veijola et al. 1998). Indeed, some psychoanalysts assert that mothers—more so than fathers—play a pivotal role in the psychosocial development of their daughters more so than sons (Chodorow 1999).

Early-life poverty was a particularly strong predictor of poor functioning among women and men. It was detrimental for functioning partly by depressing educational attainment and elevating negative affect in adulthood. Indeed, negative affect is a key link between at least one correlate of early economic adversity—harsh parenting—and adult health (Taylor et al. 2004). While the link through negative affect may simply reflect a disposition for negative mood, some scholars hypothesize that, to the extent that childhood families are chronically dysfunctional and unsupportive, it may also reflect a biological stamp on emotion and stress-response regulation (Repetti et al. 2002). Early-life poverty also predicted metabolic (obesity, diabetes) and cardiovascular conditions

among women. This finding concurs with emerging research that early-life economic hardship is a stronger predictor of many health conditions among women compared with men, including self-reported health (O'Rand et al. 2009), heart attack risk (Hamil-Luker and O'Rand 2007), obesity (Khlat et al. 2009), diabetes (Maty et al. 2008), and metabolic syndrome (Langenberg et al. 2006; Lehman et al. 2005). The fact that these reported associations were robust to intervening mechanisms—for example, adult SES, parity, and health behaviors—may reflect inherent sex differences in biological vulnerability to conditions linked with early-life poverty. However, it is unclear whether or why women are biologically more vulnerable than men to early adversities. As stated earlier, the present study cannot unequivocally adjudicate between social and biological processes; however, it does provide some insights and weak evidence for biological imprinting because the associations between early-life poverty and the two health conditions were net of important risk factors for the conditions, and because adjusting for health-related attitudes in ancillary models did not explain the associations. To the extent that we can extract from animal studies, experimental studies with rats support sex-specific biological consequences of pre- and post-natal nutrition (Dahri et al. 1995; Moura et al. 2003). Further studies should evaluate biological indicators of metabolic function in early life as one possible way to gain leverage in understanding the extent to which poverty is linked to obesity through behaviors or biological set-points. Lastly, we should bear in mind that poverty here reflects extreme deprivation: many of these adults grew up prior to the establishment of Aid to Families with Dependent Children in 1935 and its more widespread distribution after the 1960s. Further, this measure may reflect serious

deprivation if only those adults who experienced extreme hardship as a child can recall that their family received welfare.

This study has some limitations that should be noted. First, early-life conditions were retrospectively reported. Some research has evaluated the validity and reliability of retrospective childhood data, and the results are encouraging. Childhood health appears to be reliable and valid among U.S. adults (Haas 2007). Childhood social class and father's education were reliably reported by U.S. female twins (Krieger, Okamoto, and Selby 1998). A birth cohort study in Scotland found good agreement between father's occupation reported by the mother at the time of birth and that reported by the offspring 50 years later, with similar levels of agreement for men and women (Batty et al. 2005). Furthermore, a birth cohort study in Britain in which early-life SES was provided by the mothers at the time of birth found that early-life SES had a strong association with midlife functional limitations (Guralnik et al. 2006). Another potential drawback is that functional limitations were self reported. However, self reports are a fairly accurate reflection of actual ability for men and women (Merrill et al. 1997). A study that used "objective" measures of limitations by trained nurses similarly found that childhood SES independently predicted limitations (Guralnik et al. 2006). The left-censoring of the data is also potentially problematic. Because the sample is mainly white and fairly young, mortality selection should not materially bias the findings. Lastly, while the order that the exposures were entered into the models was informed by the Disablement Framework (Verbrugge and Jette 1994) such that pathology (e.g., diabetes) typically precedes declines in functioning, declines in functioning may also initiate or exacerbate pathology.

More research is needed to validate and extend these results. Future research should also explore population heterogeneity in the processes. For instance, do the processes operate similarly for specific race/ethnic groups; for adults raised in rural versus urban environments; for adults raised in extreme circumstances such as the Great Depression; for the elderly; for other health conditions? This potential heterogeneity will need to be explored with much larger datasets. Future research should also identify the specific mechanisms linking early-life exposures and functional health. For example, which specific features of childhood poverty are most salient? Because poverty's association with functioning was net of parental education, occupation, family structure, and mother-child relationship, does it reflect early-life nutrition, pathogen exposure, hormonal set-points, or other factors? The present study provides a conceptual foundation for such future work.

Conclusions

Among U.S. adults aged 45 to 74 years in 1995, functional limitations reflected experiences across the life course, and early-life experiences appeared to anchor the gender gap in functioning. Early-life poverty predicted poor functioning among men and women by depressing educational attainment and psychosocial well-being in adulthood. Further, some early-life adversities were more consequential for women than men because: (a) adult educational attainment, which tracks parental education, was more closely linked to women's than men's functioning, (b) women's relationship with their mother in early life may have an enduring influence on negative affectivity, and (c) early-

life poverty predicted women's metabolic and cardiovascular health. This study extends prior research by developing a conceptual framework for explicating the mechanisms through which early-life conditions shape later-life health in sex/gender-specific ways, and by using that framework to identify mechanisms for further research. I conclude by offering suggestions for future research and policy. First, research on gender disparities in later-life health may benefit from incorporating early-life conditions. Second, research on the early-life origins of later-life health would benefit from considering sex and gender differences in the life course processes. Third, public health interventions to improve functioning among older adults should consider targeting childhood as a critical period. In particular, tackling childhood poverty may be especially promising for improving the functional health of the U.S. population.

Table 2.1: Distribution of Early-Life and Adult Exposures and their Associations with Functional Limitations

	Distribution				Associations		
	Men		Women		Men	All	Women
	Mean	SD[1]	Mean[2]	SD	Beta[3]	Beta	Beta
Early-Life Socioeconomic Status							
Father low education (%)	63.9		70.0		0.07		0.20**
Mother low education (%)	51.9		60.5**			0.09†	
Father low-status occupation (%)	76.9		75.6			0.11†	
Mother low-status occupation (%)	82.9		81.3		-0.04		0.18*
Blue-collar occupation (%)	23.9		25.0		0.00		0.18†
Not employed (%)	59.0		56.3		-0.05		0.18*
Perceived family income (1-7)	3.8	1.1	3.9	1.2		-0.05**	
Poverty experience (%)	7.3		6.8			0.35**	
Early-Life Psychosocial Resources							
Intact family (%)	78.6		75.1			-0.12*	
Relationship with mother (1-5)	4.2	0.8	3.7**	1.1	0.00		-0.09**
Early-Life Fair/poor Health (%)	3.4		4.7		-0.25		0.53**
Adult Socioeconomic Status							
Less than high school (%)	17.0		20.1		0.17*		0.44**
High school or some college (%)	57.1		64.5**				
College (%)	25.9		15.5**			-0.17**	
Adult Psychosocial Resources							
Married (%)	83.9		66.9**			-0.07	
Negative affect (1-5)	1.4	0.5	1.6**	0.6		0.53**	
Mastery (1-7)	5.9	0.9	5.7**	1.1		-0.09**	
Adult Health Behaviors							
Parity (0 to 5 and higher)	---	---	2.6	1.5	---	---	0.05*
Obese (%)	24.6		29.2†		0.37**		0.73**
ln(pack-years)	2.5	1.5	1.6**	1.6		0.07**	
Adult Health Conditions							
Heart trouble (%)	20.8		15.8**		0.52**		0.72**
Diabetes (%)	10.8		8.7		0.53**		0.80**
High blood pressure (%)	31.2		31.1		0.19**		0.57**
Arthritis (%)	24.3		38.9**		0.40**		0.59**
Major depressive episode (%)	7.6		12.2**			0.49**	
Maximum N	728		799		728	1,527	799

†p<0.10; *p<0.05; **p<0.01 (two-sided tests)

[1] Standard deviation.

[2] Stars indicate a statistically significant gender difference in mean exposure, controlling for age, race, and sample weights.

[3] Beta is the OLS coefficient predicting functional limitations from each exposure individually, controlling for age, race, and sample weights. If there is no gender difference in the beta, it is shown under "All." Betas for adult education are in reference to "high school or some college."

Table 2.2: OLS Coefficients Predicting Functional Limitations from Early-Life and Adult Exposures among Women

Exposure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Parent Education																		
Father low		.21*	.16†	.14†	.12	.14†	.14†	.15†	.15†	.04								
Mother low		-.05																
Parent Occupation																		
Father low			.06															
Mother low			.06															
Parent Income																		
Perceived				-.07*	-.04													
Poverty					.44**	.50**	.45**	.48**	.45**	.42**	.32*	.33*	.26*	.24†	.20	.26*	.25*	.27*
Early Psychosocial																		
Intact family							-.13											
Mom relationship								-.08**	-.07*	-.07*	-.01							
Early Health																		
Fair/poor									.25									
Adult Education																		
LTHS (vs. HS)										.31**	.28**	.28**	.23**	.19*	.23**	.23**	.23**	.23**
College (vs. HS)										-.23*	-.16†	-.15	-.14	-.14	-.12	-.11	-.08	-.15†
Adult Psychosocial																		
Married											-.01							
Negative affect											.50**	.50**	.46**	.42**	.44**	.44**	.42**	.42**
Mastery											.00							
Adult Behaviors																		
Parity												.01						
Obesity													.62**	.60**	.56**	.56**	.56**	.62**
ln(pack-years)													.04*	.03†	.04*	.04*	.04†	.04*

Continued on next page.

Table 2.2 continued.

Exposure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Adult Conditions																		
Heart trouble														.50**				
Diabetes															.43**			
Hypertension																.21**		
Arthritis																	.39**	
Depression																		.22*
Intercept	.89**	.78**	.69**	.80**	.78**	.77**	.87**	.76**	.75**	.81**	.86**	.85**	.69**	.63**	.67**	.64**	.55**	.66**
Adjusted R2	.07	.08	.08	.08	.09	.09	.09	.10	.10	.12	.22	.22	.30	.33	.31	.31	.33	.30

†p<0.10; *p<0.05; **p<0.01

Notes: N=704. All models control for age and race (white, non-white) and adjusted by the sample weights.

Table 2.3: OLS Coefficients Predicting Functional Limitations from Early-Life and Adult Exposures among Men

Exposure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Parent Education																
Father low		.05														
Mother low		-.02														
Parent Occupation																
Father low			.11													
Mother low			-.09													
Parent Income																
Perceived				-.02												
Poverty					.25*	.23†	.26*	.25*	.22†	.11						
Early Psychosocial																
Intact family						-.06										
Mom relationship							.01									
Early Health																
Fair/poor								-.26								
Adult Education																
LTHS (vs. HS)									.16†	.13						
College (vs. HS)									-.08	-.05						
Adult Psychosocial																
Married										-.12						
Negative affect										.40**	.43**	.40**	.40**	.43**	.40**	.40**
Mastery										-.05						
Adult Behaviors																
Obesity											.34**	.32**	.31**	.33**	.30**	.34**
ln(pack-years)											.06**	.05**	.06**	.06**	.06**	.06**

Continued on next page.

Table 2.3 continued.

Exposure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Adult Conditions																
Heart trouble												.38**				
Diabetes													.30**			
Hypertension														.05		
Arthritis															.24**	
Depression																.18
Intercept	.61**	.58**	.60**	.61**	.59**	.64**	.59**	.60**	.59**	.70**	.53**	.44**	.50**	.52**	.47**	.52**
Adjusted R2	.07	.07	.07	.07	.07	.07	.07	.08	.08	.16	.19	.22	.20	.19	.21	.19

†p<0.10; *p<0.05; **p<0.01

Notes: N=675. All models control for age and race (white, non-white) and adjusted by the sample weights.

Figure 2.1: Major Linkages between Early-Life Conditions and Functional Limitations among Women

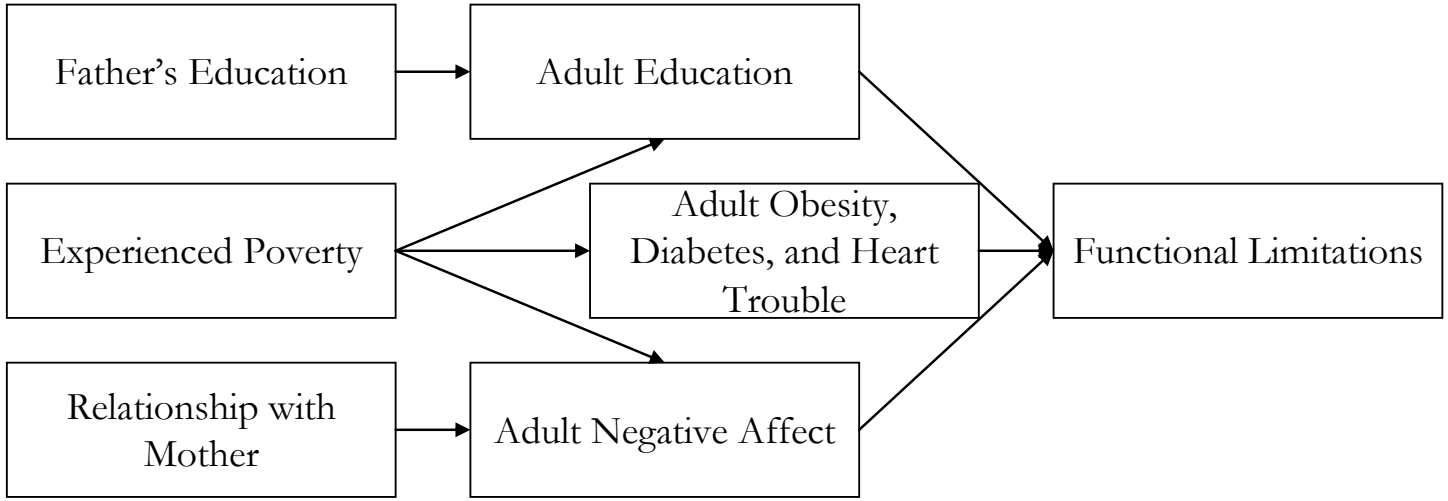
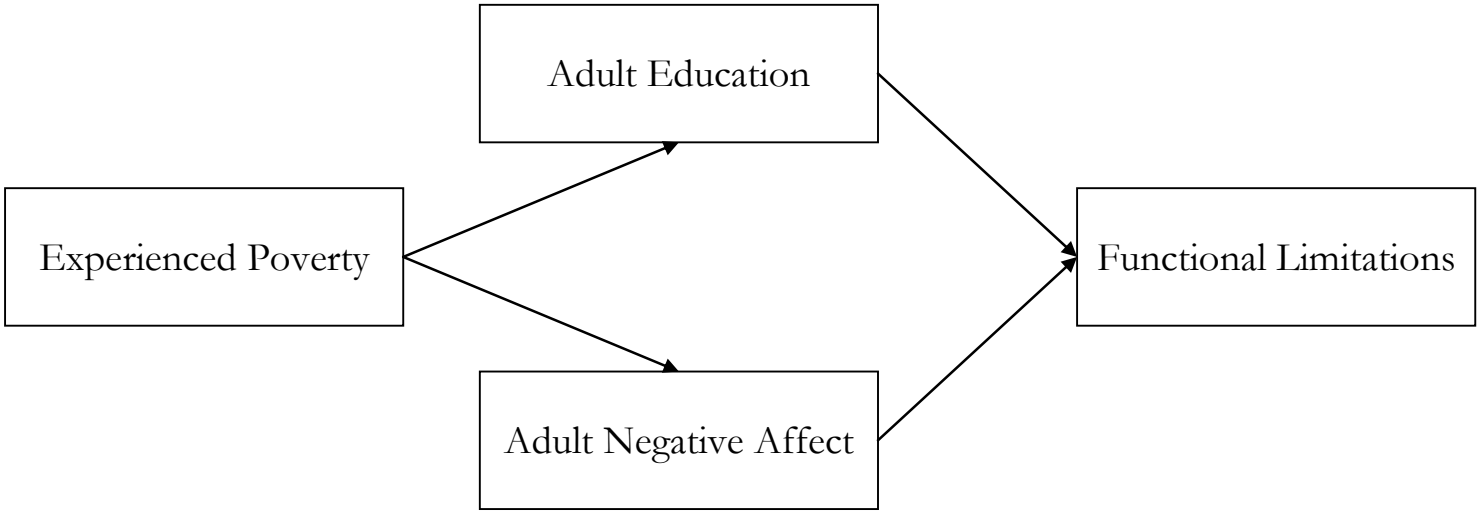


Figure 2.2: Major Linkages between Early-Life Conditions and Functional Limitations among Men



Chapter 3: The Gender Gap in Active Life Expectancy: Does Women's Longer Life in Worse Health Originate in Early Life?

CHAPTER 3 ABSTRACT

Research on women's poorer physical functioning but longer life expectancy compared with men has emphasized inequities in adult circumstances but has fallen short of fully accounting for the pattern. Recasting this research within a life-course, epidemiological framework points to the potential role of early-life conditions. This study examines the extent to which transitions in physical functioning and active life expectancy reflect early-life socioeconomic conditions for women compared with men. It uses data from the 1998 through 2008 Health and Retirement Study on adults 50 to 100 years and employs a multivariate, multistate life table approach. Having high-educated parents was important for staving off functional decline—much more so than it was at helping adults recover from it or avoid death—for women and men. Conversely, having low-educated parents increased the proportion of life spent impaired by elevating the risk of impairment somewhat more than the risk of death. Parents' education was a strong predictor of health state transitions and active life expectancy among women, and among men, but they did not predict the gaps in these outcomes between women and men, suggesting that gender disparities in these outcomes may have a stronger biological than social basis.

Research on women's poorer physical functioning but longer life expectancy compared with men has emphasized inequities in adult circumstances, such as socioeconomic resources and health behaviors, but has largely fallen short of fully accounting for the pattern. Recasting this research within a life-course, epidemiological framework points to the potential role of early-life conditions in contributing to the pattern. Early-life conditions launch long-term trajectories of social circumstances that shape functioning and longevity, and they may also impart a biological imprint on these outcomes, in ways that potentially differ for men and women. At present, we know little about whether and why early-life conditions contribute to gender gaps in functioning, mortality, and their intersection, active life expectancy. To what extent are deterioration and improvement in functioning shaped by early-life conditions for women compared with men? To what extent is active life expectancy shaped by early-life conditions for women compared with men? Do early-life conditions help explain the gender gap in these health outcomes? Do women and men who experienced adverse early-life conditions spend a greater proportion of their lives functionally impaired compared with adults from advantaged conditions, or do they spend a similar proportion impaired but within a truncated lifespan? This study addresses these questions among U.S. adults 50 to 100 years of age in the 1998 through 2008 Health and Retirement Study using a multivariate, multistate life table modeling approach.

THEORY AND EVIDENCE

Women can expect to live longer than men but experience worse functional health. In the United States in 2004, life expectancy at birth for women was 80.4 years compared with 75.2 years for men (Arias 2007). However, women are more likely than men to be functionally impaired. For example, 24.4 percent of women and 19.1 percent of men 18 years and older reported some degree of impairment such as functional limitations, difficulty performing instrumental activities of daily living (IADL) such as using a telephone, or difficulty performing basic activities of daily living (ADL) such as bathing (Centers for Disease Control and Prevention 2009). The gaps become even more pronounced with age (Gorman and Read 2006).

The combined implication of disparities in functioning and longevity can be summarized by active life expectancy (Crimmins, Hayward, and Saito 1996). Active life expectancy is defined here as the total number of years that individuals can expect to live without functional impairment and thus reflects the interplay of functional decline, functional improvement, and death. Studies of active life expectancy consistently find that, compared with men, women live a greater number of years without functional impairment, a greater number of years with impairment, and a greater proportion of life impaired (Crimmins et al. 1996; Robine and Ritchie 1991). For instance, among U.S. adults 70 years of age in the Longitudinal Study of Aging, women could expect to live an additional 11.1 years (8.9 years for men) with few to no functional limitations and 2.8 years (1.4 years for men) with IADL or ADL disability, such that the proportion of years

of life spent disabled after age 70 was 20.1 percent for women and 13.6 percent for men (Crimmins et al. 1996).

Consistent with the above definition, the gender gap in active life expectancy can be thought of as reflecting gender differences in: (a) age-specific rates of death, functional decline, and functional improvement, and (b) the underlying social and biological influences on those three processes. For instance, age-specific death rates in the United States are higher for men than women due in part to certain biological factors (e.g., lower estrogen and higher testosterone levels among men) and certain social factors (e.g., heavy cigarette smoking) that elevate men's mortality risk (Waldron 2004). At the same time, women experience higher age-specific rates of functional decline and lower rates of functional improvement than men (Leveille et al. 2000; Stuck et al. 1999). Major contributing factors likely include biological differences (e.g., lower bone and lean muscle mass among women), the greater prevalence of nonfatal chronic health conditions (e.g., arthritis, depression) among women than men, and social factors that contribute to women's poorer physical functioning such as their comparatively disadvantaged socioeconomic resources.

Indeed, the association between adult socioeconomic resources and functional ability (Mirowsky and Ross 2003), mortality (Hummer and Lariscy 2011), and active life expectancy (Crimmins and Saito 2001) is firmly established. For instance, adults with more education enjoy a longer active life expectancy and they live a greater proportion of their life free from functional impairment compared with adults with less education (Crimmins and Saito 2001). While those associations are firmly established, a life course

epidemiological framework asserts that these health outcomes reflect more than adult socioeconomic circumstances. Instead, they likely reflect the cumulative experience of socioeconomic (and other) circumstances across the life course (Kuh and Ben-Shlomo 2004). Thus, this study examines the extent to which active life expectancy reflects early-life socioeconomic circumstances, in addition to those in adulthood, for women compared with men.

Conceptually, early-life socioeconomic conditions may shape later-life functioning and mortality through "direct" and "indirect" processes. They may directly influence these outcomes through biological imprint processes. For instance, Barker (1997) claimed that prenatal nutrition may permanently alter the structure and function of organs and tissues—particularly those associated with metabolic and cardiovascular systems. Early-life conditions may also indirectly influence these outcomes through pathway processes. For instance, early-life socioeconomic environments may launch trajectories of social, psychological, and behavioral (dis)advantages, with the more proximate adult (dis)advantages shaping health. Supporting both processes, studies in the United States (Bowen and Gonzalez 2010; Haas 2008; Haas 2007; Luo and Waite 2005; Turrell et al. 2007), Britain (Guralnik et al. 2006), and Latin America (Alvarado et al. 2007) have found that early-life socioeconomic environments predict later-life functioning, and that intervening mechanisms such as adult educational attainment and health behaviors partially mediate the associations.

Early-life socioeconomic resources are a potentially important contributor to the (seemingly paradoxical) pattern of women's greater longevity but worse functional health

for at least three reasons. First, differential access to socioeconomic resources such as money, knowledge, and power is thought to be a "fundamental cause" of health disparities (Phelan et al. 2004). Adults who have access to these resources can harness them to optimize their own health and longevity, as well as the health and longevity of their children. Second, while other early-life conditions may also exert an enduring influence on later-life health, socioeconomic conditions may be the best single indicator of a collection of related exposures, such as nutrition, psychosocial stressors, hygiene, pathogen exposure, housing structure, cognitive stimulation, and neighborhood context. Third, men and women may garner differential health benefits from these resources throughout the life course. As Macintyre and Hunt (1997:316) assert, "Biological differences in vulnerability to environmental threats and differences between men and women in the specific causes of death might plausibly lead to interactions between socio-economic factors and gender in the social patterning of health." For instance, the socioeconomic gradient for all-cause mortality risk generally appears steeper for older men than women in the United States and Britain (Macintyre and Hunt 1997), although in the United States this is due to an excess of smoking-related deaths among very low-educated, never-married men (Montez et al. 2009). In contrast, the gradient for body mass index—a precursor to morbidity and functional limitations—tends to be steeper for women (Macintyre and Hunt 1997). Likewise, interactions between sex/gender and early-life socioeconomic resources may help explain the gap in active life expectancy, as described in more detail below.

Indeed, early-life socioeconomic conditions may shape later-life functioning and mortality differently for women than men. Regarding "direct" process, early-life conditions may interact with sex differences in growth (e.g., bone development), maintenance (e.g., immune systems), and reproductive function that reflect sex-specific life history strategies during a critical and highly plastic developmental window (Decaro et al. 2010). For instance, maternal stress appears to increase the risk of fetal death among males in particular (Bruckner and Catalano 2007), and may thus may have greater consequences for men's mortality risk. Also, inadequate early-life nutrition may be more consequential for women's than men's risks of osteoporosis (Kin et al. 2007) and obesity (Ravelli et al. 1999), which then adversely impact functional ability. The association between early nutrition and osteoporosis may be stronger for women because, on average, women start life with lower bone mass than men, controlling for weight (Rupich et al. 1996), and because gender-specific norms for body weight and physical activity may exacerbate the sex difference. In addition, poor postnatal nutrition and adverse childhood socioeconomic conditions have been linked with an earlier age of menopause, net of adult confounders (Hardy and Kuh 2002, 2005), which increases the years of exposure to the risk of low bone density and osteoporosis (see Shuster et al. 2010). Regarding "indirect" processes, early-life socioeconomic conditions may have differential implications for social circumstances in adulthood. For instance, early-life socioeconomic disadvantage may be more consequential for women than men if women are less able to escape it through their own social mobility.

Only a handful of studies have examined differences in the strength of the association between early-life conditions and adult functioning between men and women, and the results are mixed. Some research finds that low socioeconomic status (cf. Luo and Waite 2005; Turrell et al. 2007), poor health (Luo and Waite 2005), and poor nutrition (Alvarado et al. 2007) in early life are more strongly related to women's than men's functioning. However, there remains considerable ambiguity about whether, why, and which early-life conditions shape functioning and longevity, and whether the linkages truly differ between men and women.

Lastly, while mounting evidence supports the notion that early-life conditions predict adult functional ability—whether measured by functional limitations (Alvarado et al. 2007; Guralnik et al. 2006; Haas 2008; Haas and Rohlfsen 2010; Luo and Waite 2005; Turrell et al. 2007) or disability (Bowen and Gonzalez 2010; Freedman et al. 2008; Haas 2007)—and mortality risk (Barker 1997; Davey Smith et al. 1998; Finch and Crimmins 2004; Hayward and Gorman 2004; Kuh et al. 2002; Montez and Hayward 2011; Turrell et al. 2007; Warner and Hayward 2006), when examined as distinct outcomes, we know little about whether they predict: (a) functional ability *across the disablement process*, and (b) their *combined outcome*, active life expectancy, let alone whether they differentially shape these outcomes for men and women. For instance, prior studies have examined the relationships between early-life conditions and being in one particular state of functioning within the disablement process (e.g., no functional impairment, functional limitations, IADL, or ADL) or mortality risk. Thus, we know little about whether early-life conditions influence transitions between functioning states across the disablement

process, transitions between functioning states and death, active life expectancy, or the compression of functional impairment within the adult life span.

This study responds to the gaps outlined above and investigates the extent to which functioning, mortality, active life expectancy, and the proportion of life spent functionally impaired reflect early-life socioeconomic conditions, net of adult socioeconomic conditions, and whether the associations differ between men and women. It moves beyond all prior examinations of the role of early-life conditions on later-life functioning and longevity by examining: (a) the intersection of these outcomes as measured by active life expectancy and the proportion of life spent free from functional impairment, and (b) functional ability across multiple states within the disablement process including no functional impairment, functional limitations, difficulty with IADL, and difficulty with ADL. Specifically, this study addresses the following questions.

1. To what extent are transitions between functioning states within the disablement process related to parents' education and how do the associations differ between men and women?
2. To what extent is active life expectancy related to parents' education and how does the association differ between men and women?
3. Does adjusting for parents' education help explain the gender gap in functioning across the disablement process and in active life expectancy?
4. To what extent is the proportion of life spent functionally impaired related to parents' education and how does this differ for men and women? Do subgroups with highly-educated parents exhibit a compression of functional impairment within the life span?

5. To what extent does own educational attainment mediate the association between parents' education and the above health outcomes?

In this chapter, early-life socioeconomic conditions are measured by a summary indicator of parents' education level, while adult socioeconomic conditions are measured by their own educational attainment. While education is just one indicator of the multidimensional socioeconomic environment, a focus on "life course education" provides a parsimonious examination of how one of the major pillars of the U.S. social stratification system—educational attainment and its intergenerational transmission—shapes longevity and the quality of life in adulthood. The strengths and weakness of these measures are discussed in more detail below.

DATA AND METHODS

Data

The data come from the Health and Retirement Study (HRS), which is a household panel survey designed to study retirement processes, economic well-being, and health among U.S. adults 50 years of age and older (HRS 2008). The present study uses the RAND HRS Version J Data File, which is a cleaned and consolidated file of all 1992 through 2008 survey waves developed by the RAND Center for the Study of Aging and supported by the National Institute on Aging and the Social Security Administration (RAND 2010). The RAND file contains 30,548 adults who are representative of all cohorts born between 1890 and 1953 and their spouses. The present study begins with the

fourth wave (1998) because of inconsistencies in the physical functioning questions and their skip patterns in earlier waves. The analytic sample for the present study includes U.S.-born, non-Hispanic whites and non-Hispanic blacks 50 to 100 years of age.

Vital Status

The HRS provides vital status information from two sources. One source is the HRS Tracker File 2008, Final Version 1.0. The tracker file reports vital status at each interview wave based on information gathered during the interview process (e.g., if a spouse reported that a study member died since the last interview, the adult is classified as deceased). Month and year of death are then ascertained through an exit interview with the spouse or other knowledgeable individual. The second source of vital status information is the National Death Index (NDI), which is a computerized database of all certified deaths in the United States since 1979. The HRS provides information to the NDI on adults whose vital status was unconfirmed or presumed dead, and then vital status and date of death are ascertained by the NDI through a probabilistic matching algorithm (Lochner et al. 2008; National Center for Health Statistics 2009). For the present study, an adult is considered deceased if either source classified the adult as deceased.

Functioning States

Adults who were alive at an interview wave are classified here into one of four mutually exclusive and exhaustive functioning states (Crimmins, Hayward, and Saito 1994; Crimmins et al. 1996; Nagi 1976; Verbrugge and Jette 1994) based on a series of

questions about physical functioning. Distinguishing four functioning states provides a more fine-grained assessment of the stage(s) within the disablement process where early-life conditions may influence functional ability and hence a better understanding of the etiology of functioning. Adults were asked whether they had difficulty with certain activities because of a health or memory problem, excluding difficulties that they expected to last less than three months. Adults who reported difficulty with (including inability to do) at least one of six activities of daily living—walking across a room, dressing, bathing, eating, getting in and out of bed, toileting—are classified as having an ADL at that wave. Adults who did not experience any difficulties with activities of daily living, but some difficulty with (or unable to do) at least one of five instrumental activities of daily living—using a telephone, managing money, taking medications, shopping for groceries, preparing meals—are classified as having an IADL at that wave. Adults who did not experience difficulty with any of the eleven activities listed above, but reported some difficulty with at least one of eleven functions—walking one block, walking several blocks, sitting for two hours, getting up from a chair after sitting for long periods, climbing several flights of stairs without resting, climbing one flight of stairs without resting, stooping/kneeling/crouching, lifting or carrying weights over 10 pounds, picking up a dime from a table, reaching arms above shoulder level, pushing or pulling large objects—are classified as having a functional limitation, FL, at that wave. Adults reporting no functional difficulties of any kind are classified as healthy at that wave. The four functioning states, death, and the 16 potential transitions between states are depicted in Figure 3.1.

Education, Age, and Race

As stated earlier, the two main predictors of interest are the socioeconomic environments in early life and adulthood. The early-life environment is measured here by three binary variables that summarize both parents' education: both parents had less than eight years of education, exactly one parent had less than eight years of education, neither parent had less than eight years of education (omitted reference). The distinction at eight years reflects the fact that this is the only level of detail available in some survey waves. Missing data for mother's and father's education was imputed as less than eight years, consistent with other studies that found adults in the HRS who were missing information on father's (mother's) education level were similar on other economic and health variables to adults who reported their father (mother) had less than eight years of education (Luo and Waite 2005; Montez and Hayward 2011). The decision to use an aggregate of both parents' education was based on several factors. First, it provides a more inclusive measure of early-life "household education" compared with one parent's education. Indeed, the education level of each parent may uniquely contribute to their offspring's health, and through unique mechanisms (e.g., Case, Fertig, and Paxson 2005). Second, it avoids multicollinearity issues when using each parent's education individually, which is a particular concern when education is dichotomized. Adult's educational attainment is measured with three binary variables indicating less than a high school diploma, a high school diploma or GED, or at least some college (omitted reference), which is a good functional form for the relationship between education and all-cause mortality risk in the HRS (Brown et al. forthcoming). Lastly, all analyses were

adjusted for age and race/ethnicity. Age is a time-varying, continuous variable from 50 to 100 years. Race/ethnicity indicates non-Hispanic white (hereafter white) or non-Hispanic black (hereafter black).

Analytic Strategy

The analysis is based on a person-year file in which each individual is aged by one year beginning with their first interview until their year of death or 2008 if they survived the follow-up period. Each person-year record indicates the health state at the beginning and end of the one-year interval. Recall that the HRS data are actually collected in two-year intervals. To create a person-year file from the two-year interval data simply requires that the data structure reflect the standard assumption that all health state transitions occur in the middle of the interval (Crimmins et al. 1994, 1996). This person-year file structure correctly accounts for exposures and produces central rates. The pooling of person-year records across the HRS waves assumes a Markov process and no period effects. The total numbers of person-year records for each of the 16 potential transitions (and non-transitions) are shown in Table 3.1. Table 3.2 contains pertinent sociodemographic information.

Transition Rates between Health States

The analysis is conducted in two steps. First, health state transition rates are estimated from 16 hazard models reflecting the 16 potential health state transitions. The transition rate is defined by equation 1.0 where P_{ij} is the probability that a transition from

state i (e.g., IADL) to state j (e.g., ADL) occurs in the age interval x to x + n (for this study, n=1 year), given that the adult was in state i at age x.

$$\mu_{ij}(x) = \lim \frac{P_{ij}(x,n)}{n} as\, n \to 0 \qquad [1.0]$$

The transition rates are estimated from multivariate hazard models using PROC LIFEREG in SAS 9.2 which assume the variation in transition times between states can be described by an exponential distribution. The general form of the hazard models is provided by equation 2.0, although the models are estimated first without adult education. All models are estimated separately for men and women and adjusted by the sample weights.

$$\begin{aligned} \ln \mu_{ij}(x) = {} & \beta_{ij0} + \beta_{ij1}(age) + \beta_{ij2}(black) + \\ & \beta_{ij3}(two\ low\text{–}\ educated\ parents) + \beta_{ij4}(one\ low - educated\ parent) + \\ & \beta_{ij5}(less\ than\ high\ school) + \beta_{ij6}(high\ school) \end{aligned} \qquad [2.0]$$

In preliminary analyses, interactions were estimated between parents' education (collapsed into an "at least one low versus none low" binary variable to boost cell sizes) and adult education. Of the 32 interactions, just five were significant at $p < 0.05$ or better. Given the small number of significant interactions and no clear interpretation of them as a whole, they were dropped from the models.

The β_{ij3} and β_{ij4} coefficients indicate the degree to which parents' education predicts transition rates, net of adult education, separately for women and men. Differences between men and women in the degree to which having two low-educated

parents predicts transition rates are tested with equation 3.0, where $\beta_{ij3,women}$ and $\beta_{ij3,men}$ are the coefficients for having two low-educated parents' estimated from the gender-stratified models, while $s^2_{\beta ij3,women}$ and $s^2_{\beta ij3,men}$ are the standard errors of those coefficients.

$$z = \frac{(\beta_{ij3,women} - \beta_{ij3,men})}{\sqrt{s^2_{\beta ij3,women} + s^2_{\beta ij3,men}}} \qquad [3.0]$$

Next, the extent to which parents' education and own education contribute to the gender gap in the 16 transition rates are estimated with multivariate hazard models that aggregate men and women.

Life Table Estimates of Active Life Expectancy

The next step of the analysis employs the gender-stratified matrix of transition rates generated above to estimate total and active life expectancy using population-based, multistate life tables. These tables distribute the radix population according to the observed prevalence in each health state at age 50 in the HRS sample, and then estimate total life expectancy, life expectancy in each health state, and the health status of the life table population at each age. A detailed description of the procedures for estimating multistate life tables are available elsewhere (e.g., Crimmins et al. 1994). In the current analysis, multistate life tables are estimated separately for women and men across nine "parents' education by own education" subgroups.

RESULTS

Transition Rates between Health States

Table 3.3 (for women) and Table 3.4 (for men) examine the extent to which transition rates between health states relate to parents' education in model 1, and to parents' education net of adult education in model 2. Each row contains the antilog of hazard model coefficients for the 16 possible transitions. The rows are grouped into transitions reflecting improvement in functioning, deterioration in functioning, and death. Model 1 reveals that parents' education was most clearly predictive of deterioration in function for women and men. To illustrate, the annual risk of transitioning from a healthy state to functional limitations was 23.7 (10.7) percent greater for women (men) with two low-educated parents compared to women (men) with two high-educated parents. Further, parents' education predicted functional decline across the disablement process—for example, in early stages from healthy to limitations, and in advanced stages from difficulty with IADL to ADL. Interestingly, while deterioration rates were elevated among adults with one or two low-educated parents, improvement rates were depressed more clearly among adults with two low-educated parents. Lastly, transitions to death were only loosely linked to parents' education. However, as shown in the last row of Tables 3.3 and 3.4, when ignoring the preceding functional state, parents' education was a highly significant predictor of mortality risk among men and women, particularly among adults with two low-educated parents.

In the same tables, model 2 adjusts for adult education level. This adjustment attenuated many associations between parents' education and the transition rates; however, parents' education remained a statistically significant predictor of most transitions, particular transitions reflecting deterioration in functioning. Again, deterioration rates were elevated among adults with one or two low-educated parents, while a more exclusive subgroup—adults with two low-educated parents—exhibited depressed improvement rates. The z-scores in the last column indicate whether the strength of the association between having had two low-educated parents and each of the 16 transition rates differed between men and women. In general, the associations were similar for men and women. There were just two exceptions: having two low-educated parents elevated women's risk of transitioning from healthy to functional limitations more than it did among men (z=-2.507), while it elevated men's risk of transitioning from a healthy state to ADL disability more than it did among women (z=2.026).

In model 2, transitions to death were loosely tied to parents' education, especially among women. Two transitions are noteworthy, however. For comparative purposes, the bottom row of Table 3.3 and 3.4 shows transition rates when ignoring the preceding functional state: adults with two low-educated parents exhibited a higher risk of death, although the coefficient is significant only for men. Once the preceding functional state is taken into account, parents' education remained unrelated to the risk of death among women who were functionally impaired; however, healthy women had a 40.1 percent lower risk of death if they had two low-educated parents compared to healthy women with two high-educated parents. At first glance, this finding is peculiar. However, it

would be expected if early-life socioeconomic advantage postponed and compressed—if not altogether avoided—impairment into a later period of the life span. In other words, compared to women with two low-educated parents, women with two high-educated parents had a marginally greater risk of transitioning from healthy to dead because they were less likely experience an interim period of impairment, rather than because they were more likely to die at a given age. Table 3.4 reveals a similar but nonsignificant pattern among men. Also noteworthy is that the transition rate from IADL disability to death was marginally greater for men than women with two low-educated parents (z=1.630), as was the transition from any state to death (z = 1.716).

The information in Tables 3.3 and 3.4 is depicted in Figure 3.2. It illustrates the degree to which accounting for parents' education shapes the survival curve and its underlying health state distribution. Specifically, it compares the survival curves of high school educated adults with two low-educated parents (shaded regions) versus high school educated adults with two high-educated parents (bold lined regions). For women, although having had two high-educated parents marginally increased overall survival it more clearly postponed (i.e., compressed) impairment into a smaller portion of the life span. For men, having two high-educated parents increased overall survival and similarly postponed impairment such that there was little change in the compression of life spent impaired. That being said, the differences between the survival curves for women and men were slight and should not be overstated.

In sum, health state transitions—particularly those reflecting functional decline—were strongly associated with parents' education, net of own education, and the

associations were fairly similar between women and men. Thus, the gender gap in functioning and mortality (and by extension, active life expectancy) does not appear to reflect gender-specific responses to parents' education levels. Table 3.5, which estimates transition rates among the aggregated sample of men and women, confirms this by showing very little mediation of the male coefficient on health state transition rates when adjusting for parents' education, own education, and both education measures. In ancillary analyses, a "male by two low-educated parents" interaction was included for the three transitions with significant gender differences in the associations identified in Tables 3.3 and 3.4. Only for the transition rate from IADL to death was the male coefficient mediated to nonsignificance.

Active Life Expectancy and the Proportion of Life Spent Inactive

Table 3.6 shows the degree to which total and active life expectancy, and the proportion of life spent inactive after 65, reflect combinations of parents' and own education. Active life at 65 is defined here as the number of years spent healthy or with functional limitations, while inactive life refers to years spent with difficulty in IADL or ADL. Note the substantial variation across the nine combinations. For example, women with two low-educated parents and less than a high school degree themselves could expect to live just 17.44 years after age 65 and spend one-half (49.43 percent) of those years inactive, whereas women with two high-educated parents and at least some college education themselves could expect to live 21.60 more years with less than one-third (30.32 percent) of those years inactive. Compared with having two low-educated parents,

having two-high educated parents was worth an additional 1.8 to 2.0 years of active life for women, and an additional 2.3 to 2.5 years for men, depending on one's own education level. Figure 3.3 summarizes key information from Table 3.6 by illustrating total expected life at age 65, and the percent of life impaired after age 65, for the nine combinations of parents' and own education.

DISCUSSION

This chapter aimed to further explain the gender gap in active life expectancy by exploring the potential role of early-life socioeconomic environments, based on the hypothesis that those environments have sex/gender-specific implications for active life expectancy through differential biological imprinting and/or differential implications for adult socioeconomic conditions. To that end, it examined the degree to which transitions in functional ability across the disablement process, active life expectancy, and the proportion of life spent impaired reflected parents' education level among U.S. adults 50 to 100 years of age in the 1998 through 2008 Health and Retirement Study. This study extends prior research on the gender gap in these health outcomes by incorporating early-life conditions, and it extends prior research on the role of early-life socioeconomic conditions on functioning and mortality by: (a) integrating these outcomes into active life expectancy, (b) examining transitions across multiple states within the disablement process, and (c) evaluating the implications for compression of functional impairment within the life span.

The study reveals several novel findings. First, transitions in functional ability partly reflect the residue of early-life socioeconomic conditions, measured here as parents' education, for women and men. Parents' education most strongly predicted transitions reflecting functional decline—much more so than for transitions reflecting functional improvement or death. Freedman and colleagues (2008) reached a similar conclusion in their study of trends in ADL disability among the elderly U.S. population. They found that improvements in early-life factors over time (mothers' education in particular) contributed to declines in ADL disability from 1995 to 2006 by delaying onset rather than enhancing recovery. However, the current study reveals an important nuance—parents' education may indeed influence recovery rates, but primarily at extreme levels of deprivation, indicated here by having two low-educated parents. Taken together, these patterns provide some clues about how parents' education shapes their offspring's health and longevity: having high-educated parents was much more important for staving off functional decline than it was at helping adults bounce back from impairment. This finding is consistent with the notion that a frailty or morbidity phenotype forms in early life (Finch and Crimmins 2004). In other words, early-life exposures may leave an indelible stamp on multiple physiologic systems, creating a susceptibility to accelerated aging, frailty, reduced physiologic reserves, dysregulation, and pathology across multiple systems. That being said, the current study did not seek to identify explanatory mechanisms other than own educational attainment, so these hypotheses are offered simply to spur further research.

Second, there were few differences between men and women in the degree to which parents' education predicted health state transitions. In fact, there were no differences among transitions that involved recovery. The two differences among transitions involving deterioration were directionally mixed. Among adults in a healthy state, having two low-educated parents elevated the rate of transitioning to functional limitations more among women than men, and of transitioning to ADL disability more among men. In addition, having two low-educated parents' increased the risk of death marginally more for men, especially for men with IADL disability.

In essence, early-life socioeconomic conditions (measured by parents' education) were a strong predictor of health state transitions and active life expectancy among women, and among men, but, contrary to this study's hypotheses, they did not predict gaps in these outcomes between women and men. A recent study similarly reported that gender gaps in three measures of biological aging (inflammation, metabolic syndrome, allostatic load) across the adult life course were largely robust to inclusion of a wide range of social and behavioral exposures (and their interactions with gender), leading the authors to posit that gender differences in health and aging trajectories may have a much stronger biological than social basis (Yang and Kozloski 2011). The authors further speculated that endogenous differences in these biological aging processes may underlie the higher prevalence of functional impairment among women. That study, combined with the current findings, suggest that socioeconomic conditions across the life course may make only a marginal contribution to the gender gap in physical functioning. Given that inequities in socioeconomic conditions: (a) are a "fundamental cause" of disparities

in preventable health outcomes, (b) strongly correlate with a wide range of social and behavioral exposures, yet (c) did not contribute to the gender gap in functional ability in this study, a greater focus on the endogenous, biological basis of gender differences in physical functioning may be in order, consistent with a recent recommendation by the Institute of Medicine (Institute of Medicine 2001). This is not to suggest that gender gaps in all health conditions are insensitive to early-life socioeconomic conditions. For instance, they may exhibit a stronger association with metabolic (Heraclides et al. 2008; Khlat et al. 2009; Langenberg et al. 2006; Lehman et al. 2005; Maty et al. 2008) and cardiovascular conditions (Galobardes et al. 2006; Hamil-Luker and O'Rand 2007) among adult women than men. Other measures of socioeconomic conditions should also be evaluated.

Third, active life expectancy reflected "life course socioeconomic status," measured by combinations of parents' and adults' education, and the disparities between subgroups were striking. For instance, women with two low-educated parents and who themselves did not graduate from high school could expect to live just 17.44 years beyond age 65 with almost one-half of those years spent inactive. In contrast, women with two high-educated parents and who themselves had achieved at least some college education could expect to live an additional 21.60 years with less than one-third of those years spent inactive. Being raised in favorable early-life socioeconomic conditions increased total and active life expectancy, net of own educational attainment, for women and men. Compared with having two low-educated parents, having two-high educated

parents was worth an additional 1.8 to 2.0 years of active life for women, and an additional 2.3 to 2.5 for men, depending on one's own level of education.

Fourth, while experiencing favorable early-life socioeconomic conditions postponed functional impairment into a later portion of the life span for women and men, because men also experienced a marginally greater postponement of death from advantaged early-life conditions than women (e.g., 1.40 to 1.65 additional total years for men depending on their own education level, versus 0.42 to 0.59 years for women), men experienced less compression of functional impairment within the life span than did women. That being said, the differences between the survival curves for women and men were slight and should not be overstated.

The focus on education as the sole indicator of early and adult socioeconomic conditions has strengths and weaknesses, conceptually and empirically. Conceptually, it provides for a clear and parsimonious focus on one of the major pillars of the U.S. social stratification system. Empirically, one of the main strengths of adult education is that it tends to be more stable than other measures of socioeconomic status such as occupation, income, or wealth, and it is less prone to reverse causality. In addition, educational attainment may be more relevant than other measures of socioeconomic status for adults who have retired from the workforce, are currently unemployed, or are out of the labor force, which is particularly important when comparing subgroups with disparate occupational histories. Further, educational attainment is generally prior to income and occupation in the causal sense (Mirowsky and Ross 2003). Parents' education is a strong predictor of numerous health outcomes and the association often holds even when

statistically adjusting for parents' occupation and household income (Luo and Waite 2005). Incorporating additional indicators of socioeconomic status may provide a broader measure; however, the additional complexity may not outweigh the loss of parsimony, particularly if the additional indicators are simply downstream mediators of education.

Another potential limitation of this study is that functional ability was self-reported, although self-reports are a fairly accurate reflection of actual ability for men and women (Merrill et al. 1997). Another consideration is that this study focused on the role of early life on a portion of the disablement process—that is, only after precipitating pathology or impairment manifests itself as a functional deficit. Thus, to move closer to the "root causes" and pathways between early-life conditions and adult functioning, future studies should integrate earlier stages of the disablement process—for example, before pathology or impairment has manifested in functional deficits. Biological indicators of preclinical conditions, measures of preclinical mobility disability (Gregory et al. 2011), and measures of frailty (e.g., strength, balance) (Fried et al. 2004) may be especially useful to that end. Future studies may want to examine race differences, although prior studies using the HRS have not found black-white differences in how early-life socioeconomic status relates to functioning (Haas and Rohlfsen 2010; Luo and Waite 2005). Lastly, these findings should not be extrapolated to other ages, cohorts, or geographical regions as the results are likely moderated by contextual factors such as the epidemiological, political, and social environments. For example, one potential explanation for the shift in the survival curve being more pronounced among men here is that women may simply be optimizing their longevity under existing circumstances.

Conclusions

Active life expectancy among adults 50 to 100 years of age in the 1998 through 2008 Health and Retirement Study reflected socioeconomic conditions in early life and adulthood, measured by parents' and own educational attainment, respectively. Having high-educated parents was important for staving off functional impairment—much more so than it was at helping individuals recover from it or avoid death—for women and men. Conversely, having low-educated parents increased the proportion of life spent functionally impaired by elevating the risk of impairment somewhat more than the risk of death. Parents' education was a strong predictor of health state transitions and active life expectancy among women, and among men, but, contrary to this study's hypotheses, they did not predict the gaps in these outcomes between women and men. Because parents' education predicted these outcomes, net of own educational attainment, early-life socioeconomic exposures may leave an indelible stamp on multiple physiologic systems and create a general susceptibility to accelerated aging, frailty, reduced physiologic reserves, and pathology across multiple systems. Policy efforts to improve the functional health and quality of life among the U.S. population may benefit from investments in education and childhood circumstances.

Figure 3.1: Health States and Potential Transitions across States and Death

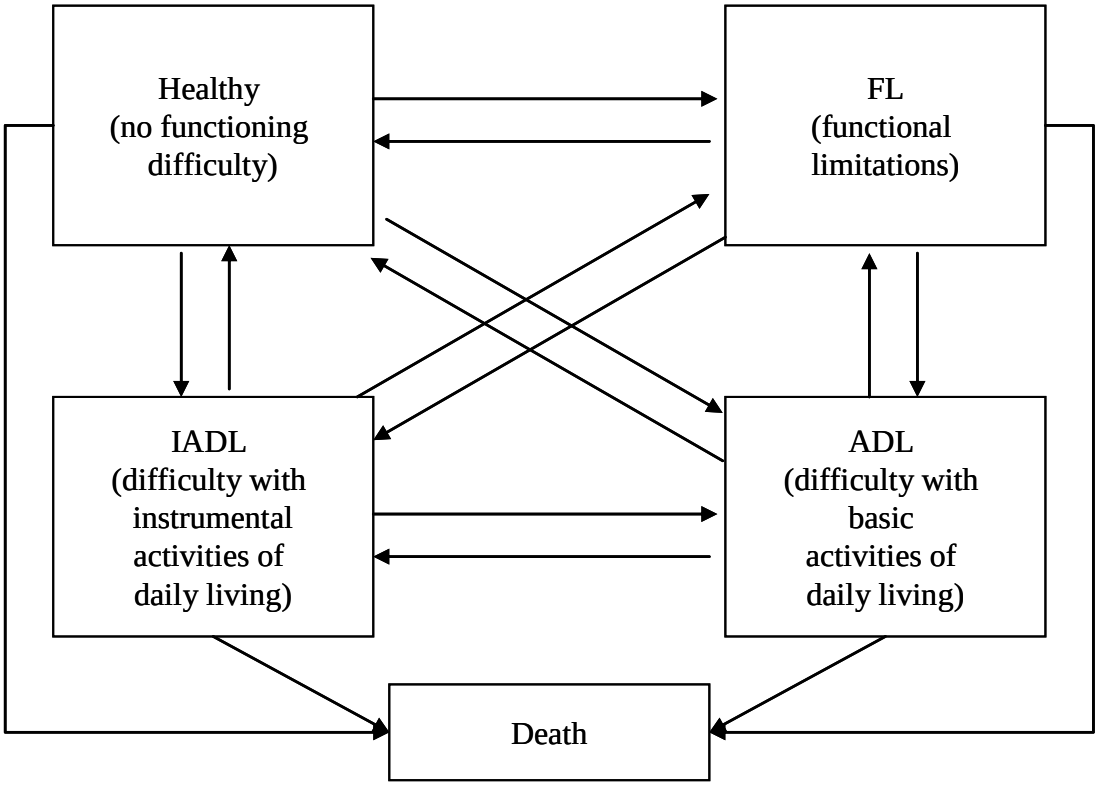


Table 3.1: Unweighted Distribution of Person-Year Records by Health State at the Beginning and End of Each Person-Year Interval for Men (and Women)

	State at End of Interval					
State at Beginning of Interval	Healthy	FL	IADL	ADL	Death	Total
Healthy	19,738 (18,203)	3,310 (3,831)	312 (198)	301 (304)	216 (110)	23,877 (22,646)
FL	2,453 (2,899)	22,569 (37,102)	669 (863)	1,610 (2,652)	621 (488)	27,922 (44,004)
IADL	181 (95)	489 (529)	2,121 (2,588)	388 (769)	185 (202)	3,364 (4,183)
ADL	110 (99)	997 (1,743)	254 (538)	6,980 (14,009)	845 (1,275)	8,386 (17,664)
Total	22,482 (21,296)	26,565 (43,205)	3,356 (4,187)	9,279 (17,734)	1,867 (2,075)	63,549 (88,497)

Notes: "Healthy" refers to no functional impairment, FL refers to functional limitations, IADL refers to difficulty with instrumental activities of daily living, and ADL refers to difficulty with basis activities of daily living. See the methods section for a more detailed explanation.

Table 3.2: Weighted Distribution of Person-Year Records among Men and Women

	Men	Women
Age (years)	65.3	66.6
Race/ethnicity (%)		
Non-Hispanic White	91.1	89.3
Non-Hispanic Black	8.9	10.7
Parents' Educational Attainment (%)		
Both parents had less than 8 years education	19.6	22.2
One parent had less than 8 years of education	19.3	20.2
Neither parent had less than 8 years of education	61.1	57.6
Adult Educational Attainment (%)		
Less than high school	17.5	18.2
High school	32.9	40.0
More than high school	49.5	41.7
Combinations of Parent's, Adult Education (%)		
Both low, less than high school	8.3	9.6
Both low, high school	6.9	8.6
Both low, more than high school	4.4	3.9
One low, less than high school	4.2	3.9
One low, high school	7.4	9.6
One low, more than high school	7.7	6.8
Both high, less than high school	5.0	4.7
Both high, high school	18.7	21.9
Both high, more than high school	37.4	31.0
Unweighted Number of Person-year Records	64,349	88,497

Table 3.3: Antilogs of Regression Coefficients Predicting Transition Rates between Health States among Women

		Model 1		Model 2				
		Parents' Education[1]		Parents' Education[2]		Adult's Education[1]		
State at Start	State at End	Both low	One low	Both low	One low	Less than HS	HS	z-score[2]
Improvement								
FL	Healthy	0.811***	0.875**	0.882*	0.919†	0.752***	0.830***	0.974
IADL	Healthy	0.508*	1.326	0.722	1.544†	0.380**	0.595*	0.412
IADL	FL	0.821†	0.834†	0.889	0.850	0.786†	0.981	-0.765
ADL	Healthy	0.702	0.906	0.927	1.012	0.406**	0.523**	-1.619
ADL	FL	0.771***	0.917	0.854*	0.947	0.728***	0.919	1.419
ADL	IADL	0.937	1.090	0.858	1.048	1.357*	1.286*	-0.035
Deterioration								
Healthy	FL	1.237***	1.204***	1.211***	1.185***	1.031	1.173***	-2.507**
Healthy	IADL	1.353	1.823***	1.204	1.784***	1.537*	0.971	0.925
Healthy	ADL	1.387*	1.430**	1.237	1.394**	1.518*	1.135	2.026*
FL	IADL	1.409***	1.410***	1.294**	1.380***	1.335**	0.859†	0.573
FL	ADL	1.262***	1.200***	1.161**	1.164**	1.325***	0.963	1.472
IADL	ADL	1.216*	1.294**	1.153	1.258*	1.154	1.124	-0.107
Death								
Healthy	Dead	0.682	0.654	0.599†	0.641	1.568	1.173	0.811
FL	Dead	1.212†	1.123	1.057	1.057	1.583***	1.159	-0.127
IADL	Dead	0.958	0.782	0.906	0.772	1.137	0.954	1.630†
ADL	Dead	1.009	0.972	0.947	0.946	1.207*	1.102	1.159
Any state	*Dead*	*1.171***	*1.047*	*1.021*	*0.986*	*1.508****	*1.149**	1.716†

***p<0.001, **p<0.01, *p<0.05, †p<0.10

[1] Omitted reference for parents' education is "two high-educated parents," and for adult education is "more than high school."

[2] The z-score tests for statistically significant gender differences in the coefficient for "both low-educated parents" in model 2.

Table 3.4: Antilogs of Regression Coefficients Predicting Transition Rates between Health States among Men

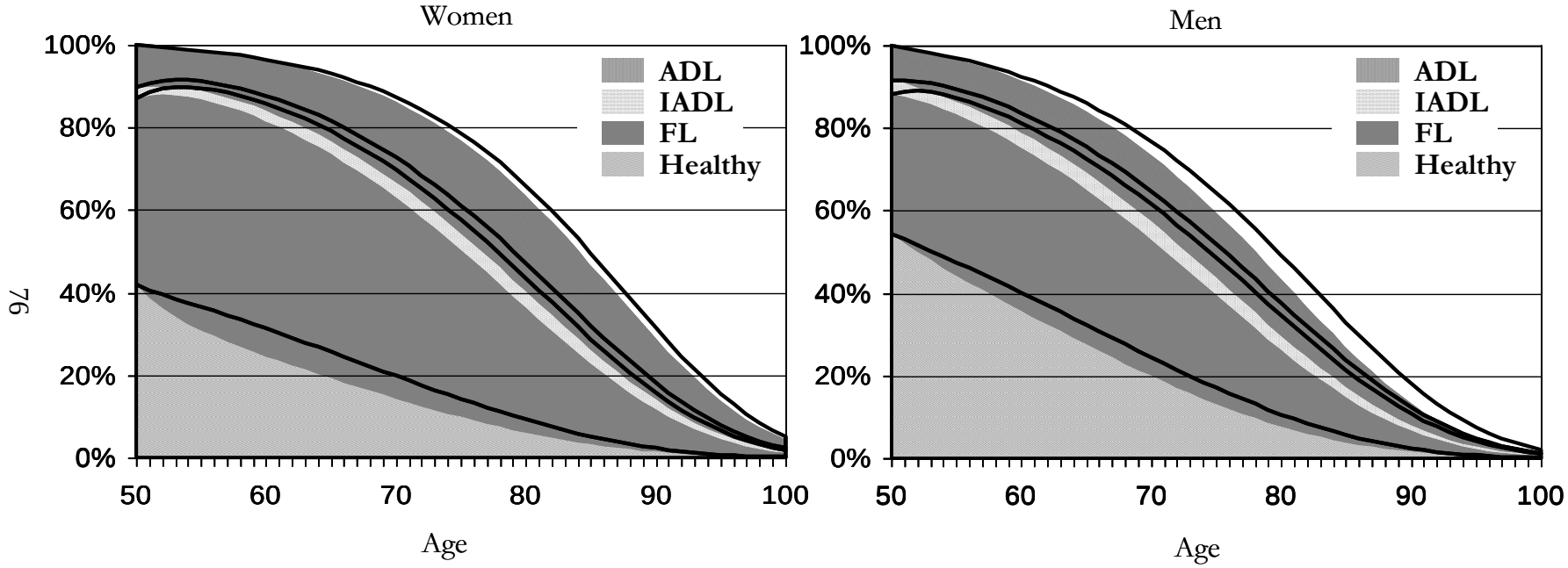
		Model 1		Model 2				
		Parents' Education[1]		Parents' Education[2]		Adult's Education[1]		
State at Start	State at End	Both low	One low	Both low	One low	Less than HS	HS	z-score[2]
Improvement								
FL	Healthy	0.864**	0.909†	0.954	0.950	0.705***	0.847***	0.974
IADL	Healthy	0.821	0.824	0.843	0.847	0.887	1.009	0.412
IADL	FL	0.777*	0.875	0.782*	0.878	0.983	0.935	-0.765
ADL	Healthy	0.460**	0.704	0.499**	0.744	0.777	0.678†	-1.619
ADL	FL	0.918	0.866†	0.991	0.889	0.795**	0.975	1.419
ADL	IADL	1.139	1.461*	1.000	1.419*	1.413*	0.786	-0.035
Deterioration								
Healthy	FL	1.107*	1.225***	1.014	1.165***	1.310***	1.266***	-2.507**
Healthy	IADL	2.070***	1.448**	1.520**	1.270	2.658***	1.278†	0.925
Healthy	ADL	2.299***	1.384*	1.920***	1.250	1.753***	1.600***	2.026*
FL	IADL	1.651***	1.351**	1.400***	1.241*	1.789***	1.289**	0.573
FL	ADL	1.400***	1.217**	1.317***	1.182**	1.243**	1.063	1.472
IADL	ADL	1.138	1.244†	1.133	1.238	1.030	0.926	-0.107
Death								
Healthy	Dead	0.936	1.004	0.793	0.906	1.671*	1.677**	0.811
FL	Dead	1.066	1.066	1.035	1.050	1.117	1.046	-0.127
IADL	Dead	1.392†	1.419†	1.427†	1.438†	0.900	0.912	1.630†
ADL	Dead	1.069	1.012	1.086	1.020	0.944	0.944	1.159
Any state	*Dead*	*1.278****	*1.143**	*1.185***	*1.099*	*1.299****	*1.197***	1.716†

***p<0.001, **p<0.01, *p<0.05, †p<0.10

[1] Omitted reference for parents' education is "two high-educated parents," and for adult education is "more than high school."

[2] The z-score tests for statistically significant gender differences in the coefficient for "both low-educated parents" in model 2.

Figure 3.2: Percent of 100,000 Radix Population Surviving across Health State by Age in the Life Table Population among Adults with a High School Diploma by Parents' Education Level



Notes: Shaded regions reflect survivorship among adults with two low-educated parents, while the bold lines reflect survivorship among adults with two high-educated parents.

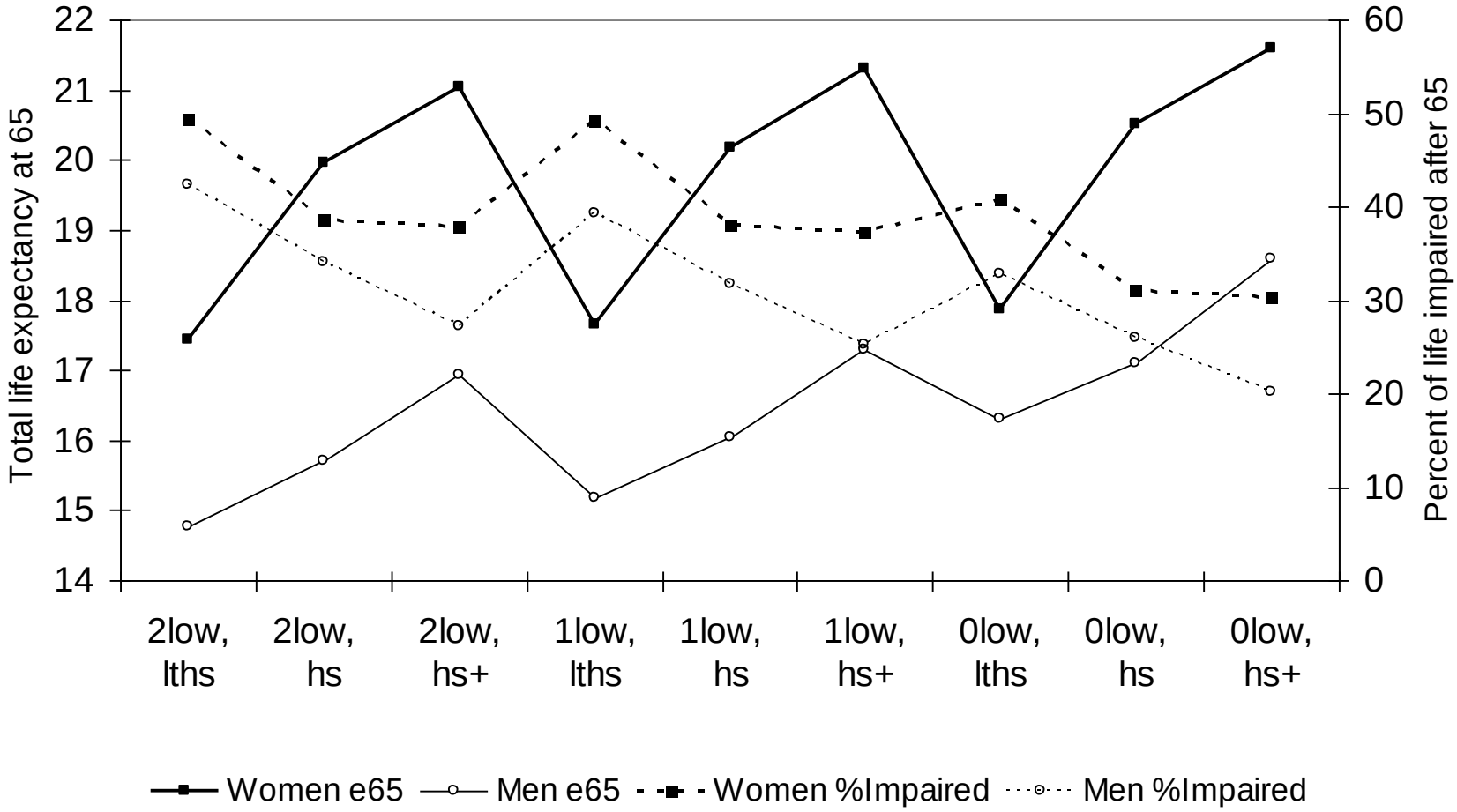
Table 3.5: Antilogs of Regression Coefficients Predicting Transition Rates between Health States among Men and Women

		Baseline	Adjusted for Parents' Education	Adjusted for Own Education	Adjusted for Parents' and Own Education
State at Start	State at End	Male	Male	Male	Male
Improvement					
FL	Healthy	1.388***	1.386***	1.377***	1.376***
IADL	Healthy	2.036***	2.059***	2.058***	2.079***
IADL	FL	1.013	1.017	1.018	1.020
ADL	Healthy	1.984***	1.975***	1.962***	1.955***
ADL	FL	1.052	1.050	1.053	1.052
ADL	IADL	0.948	0.950	0.946	0.948
Deterioration					
Healthy	FL	0.794***	0.794***	0.807***	0.806***
Healthy	IADL	1.392***	1.394***	1.400***	1.393***
Healthy	ADL	0.969	0.970	0.994	0.990
FL	IADL	1.209***	1.217***	1.194***	1.199***
FL	ADL	1.017	1.022	1.009	1.012
IADL	ADL	0.658***	0.658***	0.655***	0.656***
Death					
Healthy	Dead	1.781***	1.779***	1.839***	1.844***
FL	Dead	2.084***	2.088***	2.078***	2.080***
IADL	Dead	1.295*	1.294*	1.293*	1.294*
ADL	Dead	1.531***	1.530***	1.530***	1.529***

Table 3.6: Expected Remaining Years of Total Life, Active Life, and Inactive Life among Women and Men 65 Years of Age by Parents' Education and Own Educational Attainment

			Expected Active Life			Expected Inactive Life			
		Total Life	Total	Healthy	FL	Total	IADL	ADL	Percent Inactive
Women									
Parents' Education	*Own Education*								
Both low	Less than high school	17.44	8.82	1.92	6.90	8.62	1.31	7.32	49.43
Both low	High school	19.95	12.27	2.41	9.85	7.69	1.12	6.57	38.53
Both low	More than high school	21.04	13.07	3.42	9.65	7.97	1.20	6.77	37.90
One low	Less than high school	17.65	8.98	2.00	6.98	8.67	1.45	7.22	49.13
One low	High school	20.18	12.50	2.55	9.95	7.68	1.23	6.45	38.06
One low	More than high school	21.30	13.34	3.62	9.72	7.95	1.30	6.65	37.35
Both high	Less than high school	17.86	10.59	2.82	7.77	7.27	1.24	6.02	40.69
Both high	High school	20.52	14.15	3.44	10.71	6.37	1.03	5.34	31.04
Both high	More than high school	21.60	15.05	4.74	10.31	6.55	1.08	5.47	30.32
Men									
Parents' Education	*Own Education*								
Both low	Less than high school	14.76	8.50	2.55	5.95	6.26	1.54	4.72	42.40
Both low	High school	15.71	10.35	3.50	6.85	5.36	1.12	4.24	34.09
Both low	More than high school	16.94	12.32	5.42	6.90	4.61	0.96	3.65	27.25
One low	Less than high school	15.18	9.22	2.62	6.60	5.96	1.50	4.46	39.27
One low	High school	16.05	10.95	3.47	7.48	5.10	1.05	4.04	31.76
One low	More than high school	17.28	12.92	5.34	7.58	4.36	0.91	3.44	25.21
Both high	Less than high school	16.31	10.98	3.49	7.48	5.34	1.34	4.00	32.71
Both high	High school	17.11	12.68	4.48	8.20	4.43	0.94	3.49	25.89
Both high	More than high school	18.59	14.84	6.72	8.12	3.75	0.80	2.95	20.15

Figure 3.3: Total Expected Life at Age 65 and the Percent of Life Impaired after Age 65 by Combinations of Parents' and Own Education



Chapter 4: How do Socioeconomic Exposures Accumulate across the Life Course to Predict Functioning and Mortality by Race and Gender?

CHAPTER 4 ABSTRACT

Socioeconomic resources ("SER") in early-life and adulthood independently predict numerous health outcomes in later life. However, it is unclear whether exposures to SER accumulate: (a) additively such that the health benefits of adult SER are irrespective of early-life SER, or (b) interactively such that the benefits are contingent on early-life SER, and whether the accumulation differs between demographic subgroups. This chapter addresses these questions for functional limitations and mortality risk among adults 50 years of age and older in the 1992 through 2008 Health and Retirement Study. The accumulation differed markedly by outcome and race-gender subgroup. Among white men, SER-related exposures accumulated additively to predict functioning; however they accumulated synergistically for white women and blacks. The mortality results were nuanced. Among white men, SER-related exposures accumulated additively to predict mortality except at very low levels of SER due to an apparent ceiling effect among men with low SER in both early life and adulthood. Among white women, exposures accumulated synergistically if they achieved more than a high school diploma. Among black women, only adult SER was predictive. Among black men, neither early-life nor adult SER were significant predictors; however, the coefficients were substantively large and indicated both ceiling and floor effects.

While the link between adult socioeconomic resources ("SER") and adult health and longevity is firmly established, mounting evidence reveals a strong link between early-life socioeconomic resources and these adult outcomes. Indeed, a life course epidemiological perspective asserts that adult health and longevity reflect cumulative exposure to socioeconomic (and other) circumstances across the entire life span. Although evidence is mounting that socioeconomic resources during both early life and adulthood shape health and longevity, we know little about how cumulative exposure to these resources shapes these outcomes and whether it does so similarly across population subgroups. In other words, do exposures to socioeconomic resources in early life and adulthood independently contribute to adult health and longevity? If so, do they accumulate additively such that the health and longevity benefits of adult resources are irrespective of one's early-life resources, or do they accumulate interactively such that the benefits of adult resources are contingent on one's early-life resources? Further, how does the accumulation differ for women compared with men, and for whites compared with blacks? This study addresses these questions for non-Hispanic white and black men and women 50 years of age and older in the 1992 through 2008 Health and Retirement Study. Addressing these questions in a systematic fashion will provide insights into how these fundamental resources accumulate to shape adult health and longevity, and they can inform public health agendas by revealing the portion(s) of the life course which are most responsive to programs aimed at improving population health.

THEORY AND EVIDENCE

Adults with greater socioeconomic resources—more years of education, more income, and more favorable occupational characteristics—live longer and healthier lives than adults with fewer of these resources (e.g., Crimmins et al. 1996; Hummer and Lariscy 2011; Mirowsky and Ross 2003). In fact, differential access to SER between population subgroups may be the "fundamental cause" of preventable health disparities (Link and Phelan 2002; Phelan et al. 2004). Adults with greater access to SER have greater access to knowledge, power, money, and salubrious social ties which they can harness to optimize their health—and their children's health—through better nutrition, housing, and medical care, for example.

While the association between adult SER and adult health and longevity is firmly established—even if the mechanisms are not entirely understood—a life course epidemiological perspective asserts that these outcomes reflect SER across the entire life span. In fact, mounting evidence from the United States and many European countries indicates that adult health and longevity reflect the accumulation of physical (e.g., nutrition, infection disease) and social (e.g., education, social ties) exposures that occur during gestation, childhood, adolescence, and adulthood (Kuh and Ben-Shlomo 2004; Montez and Hayward 2011). Thus, there has been growing interest across multiple disciplines in whether, how, and why early-life SER contribute to later-life health and longevity, net of the contribution of adult SER.

Conceptually, early-life SER may shape later-life health and longevity through "direct" and "indirect" processes. They may directly influence these outcomes through biological imprint processes. For instance, geographic regions in Britain characterized by poor social conditions and high neonatal mortality rates during certain cohorts' early life experienced disproportionate death rates from coronary heart disease and stroke during those cohorts' later life, which led Barker (2007) to hypothesize that inadequate nutrition in utero permanently alters the structure and function of organs and tissues, particularly those associated with metabolic and cardiovascular systems. Corroborating this hypothesis, subsequent studies reported that birth weight (a marker of prenatal nutrition) predicts the risks of obesity (Ravelli et al. 1999) and diabetes (Barker 1997; Innes et al. 2002) in later life, particularly among women, irrespective of adult circumstances. Other socioeconomic-related resources in early life, such as chronic pathogen exposure, may also impart a biological stamp on outcomes such as mortality risk (Finch and Crimmins 2004) and decreased resistance to upper respiratory infections in adulthood (Cohen et al. 2004). In addition to direct biological imprinting, early-life SER may shape adult health and longevity through indirect, pathway processes. Specifically, early-life SER may launch trajectories of social, psychological, and behavioral (dis)advantages, with only the more proximate adult (dis)advantages shaping these health-related outcomes. For instance, Hayward and Gorman (2004) found that the strong relationship between early-life SER and all-cause mortality risk among older U.S. men was attenuated to nonsignificance (except for parents' nativity) once adult social and behavioral characteristics were statistically accounted for. Similarly, a study of U.S. blacks

concluded that their early-life SER was positively related to their late-life mortality risk primarily because it corresponded with adult SER (Preston, Hill, and Drevenstedt 1998).

Despite compelling evidence linking both early-life and adult SER with later-life health and longevity, scholars have given scant attention to systematically examining precisely how exposure to SER *accumulates* across the life course to shape these outcomes, and whether it does so similarly across population subgroups. In other words, do SER in early life and adulthood independently contribute to adult health and longevity? If so, do they accumulate: (a) additively such that the health and longevity benefits of adult SER are irrespective of one's early-life SER, or (b) interactively such that the benefits of adult SER are contingent on one's early-life SER, and (c) how does the accumulation differ between race-gender subgroups?

A major omission in this literature is an inattention to potential heterogeneity in the ways that race and gender—and their combination—may moderate how exposure to SER accumulates across the life course to shape health. This omission has likely led to overgeneralizations in these life course processes. Given that the health-related benefits of educational attainment may differ between men versus women (Elo and Preston 1996; Montez et al. 2009; Nathanson and Lopez 1987) and between whites and blacks (Kimbro et al. 2008; Lin et al. 2003), the health-related benefits of life course SER may also accumulate uniquely for these subgroups. For instance, among U.S. adults, both childhood and adult SER independently predicted women's heart attack risk, but only adult SER predicted men's risk, which the study's authors speculated partly reflects greater social mobility among men (Hamil-Luker and O'Rand 2007). Further, we know

little about whether the benefits accumulate uniquely for race-gender subgroups, as might be expected from an intersectionality approach. This approach asserts that the socially-constructed categories of race, gender, and class must be viewed as mutually constituted and interconnected, such that their health implications are best understood by considering these categories as intertwined and inseparable (Mullings and Schulz 2006). Demonstrating the value of an intersectionality approach, Warner and Brown (2011) found that U.S. black women—unlike white women or black men—exhibited a uniquely accelerated age-related disablement trajectory; and Read and Gorman (2006) similarly found that disparities across multiple health outcomes exhibited distinct patterns between race-gender groups. In contrast to the double jeopardy hypothesis which posits that the deleterious health implications of minority race (e.g., black) and gender (e.g., female) are additive in an approximately mathematical sense, an intersectionality approach makes no such assumption. In support, a 15-year panel study found that black men 50 to 64 years of age experienced the most rapid declines in self-rated health compared with white men, white women, and black women (Ferraro and Farmer 1996).

This study aims to address these gaps by evaluating competing hypotheses about how exposure to early-life and adult SER accumulates to predict two specific outcomes—functional limitations and all-cause mortality risk—in later life. This evaluation has important theoretical and practical benefits. Theoretically, it will advance our understanding of life course epidemiology by systematically examining how one of the most fundamental inputs into health accumulates across the life course. It will also provide insights into how (and potentially why) life course SER shape functioning and

longevity by indirectly examining the extent to which biological imprint and social pathway mechanisms explain the overall association. For instance, if adults who experienced disadvantaged early-life SER garner substantially fewer health benefits from higher adult SER than do adults from advantaged early-life SER, this may reflect some degree of biological imprinting in early life. Practically, the present study can inform public health investments. For instance, if the effect of adverse early-life SER on adult health cannot be ameliorated by higher SER in adulthood, then the most efficacious time period for social investments may lie in early-life environments. It will also indicate whether previous studies have been underestimating the extent of socioeconomic-related health disparities within the U.S. population by heretofore focusing on a single (i.e., adult educational attainment) indicator derived exclusively from the adult life course. In other words, to the extent that adult SER tracks early-life SER, and if both SER exposures independently contribute to adult health, previous studies have underestimated the degree of health disparities within the population.

Specifically, this study evaluates four hypotheses on how early-life SER (measured by fathers' and mothers' education) and adult SER (measured by own educational attainment) accumulate to predict functional limitations and all-cause mortality risk among non-Hispanic white and black men and women 50 years of age and older in the 1992 through 2008 Health and Retirement Study. Briefly, the first hypothesis asserts that exposures to early-life and adult SER do not, in fact, accumulate to shape functioning and mortality. Thus, either parents' education or adult educational attainment, but not both, predicts these outcomes. In the second hypothesis, the health

and longevity benefits of adult SER may simply "add to" the benefits accrued from early-life SER. For example, each year of adult educational attainment might reduce the risk of poor health by X percent, irrespective of parents' education. In the third and fourth hypotheses, the health and longevity benefits of adult SER are contingent on one's early-life SER. In the third hypothesis, the benefits of higher adult SER are most pronounced among adults from advantaged early-life SER, while in the fourth hypothesis the benefits of higher adult SER are most pronounced among adults from adverse early-life SER. These hypotheses are elaborated in more detail below.

Hypothesis 1: No Accumulation of Life Course SER

Adult functioning and mortality risk may reflect SER from a specific period of the life course. If they reflect only early-life SER, this suggests that early exposures impart a biological imprint during this critical period of development that cannot be subsequently altered. For instance, in a study of Scottish men, early-life SER, not adult SER, predicted deaths from stroke and stomach cancer (Davey Smith et al. 1998), the latter of which has been attributed in part to *Helicobacter pylori* exposure in early life within poor hygiene environments. Early-life SER may also be particularly crucial for the development of physiological and structural characteristics needed for optimal functioning. For instance, low birth weight—a correlate of SER—predicts lower lean muscle and bone mass in later life, leading the study's authors to conclude that "…bone and muscle growth may be programmed by genetic and/or environmental influences during intrauterine life" (Gale et al. 2001:267). Alternatively, early-life SER may not leave any biological residue, such

that adult SER is the sole contributor to adult functioning and longevity. In support, a study of Scottish men found that early-life SER did not exhibit any association with deaths from accidents and violence (Davey Smith et al. 1998). A study of U.S. men found that while early-life SER was a strong predictor of all-cause mortality risk, the association was largely due to transmission of SER between parents and sons (Hayward and Gorman 2004).

Hypothesis 2: Additive Accumulation of Life Course SER

Exposure to early-life and adult SER may accumulate additively to shape functioning and mortality. In other words, the incremental benefits of adult SER for these outcomes may be irrespective of early-life SER. While this hypothesis allows for adult SER to partially mediate the association between early-life SER and adult functioning and mortality, the association remains statistically significant. A negligible degree of mediation implies greater support for a biological imprint mechanism, while a higher degree of mediation implies greater support for a pathway mechanism.

Among the small handful of studies that report statistical tests for interactions between early-life and adult SER on later-life health and longevity, some find no statistically significant interaction and thus support an additive accumulation. Specifically, the accumulation appears additive for all-cause mortality risk among U.S. men (Hayward and Gorman 2004), all-cause mortality risk among British adults (Kuh et al. 2002), deaths from coronary heart disease and stroke among Scottish men (Davey Smith et al. 1998), and age at menopause (a precursor to functional decline) among

British women (Lawlor, Ebrahim, and Smith 2003). More commonly, studies do not conduct and/or report statistical tests for an interactive accumulation, yet conclude that the accumulation is additive based simply on the fact that the association between early-life SER remains when adult SER is accounted for (e.g., Guralnik et al. 2006).

Alternatively early-life and adult SER exposures may accumulative interactively to shape functioning and mortality. In other words, the health-related benefits of adult SER may be contingent on the SER the individual experienced during childhood. Scholars typically consider two hypotheses for why this might occur: Synergistic Accumulation and Catch-up Accumulation.

Hypothesis 3: Synergistic Accumulation of Life Course SER

This hypothesis asserts that the health-related benefits of adult SER will be most pronounced among adults raised in advantaged, rather than disadvantaged, early-life SER contexts. The underlying theory is that, unlike adults from disadvantaged early contexts, adults from advantaged early contexts have the requisite health stock—for example, optimal metabolic and cardiovascular systems, high bone density, high cognitive function—from which to further capitalize on in adulthood. Indirect empirical support comes from the vast sociological literature on cumulative advantage that finds the health benefits garnered from adult SER accrue and widen with age (Lynch 2003; Mirowsky and Ross 2008; Ross and Wu 1996). Panel A in Figure 4.1 illustrates how scholars typically envision this hypothesis using mortality risk as an example: the slope of

mortality risk reduction with increasing adult SER is steepest for adults who were raised with advantaged childhood SER.

However, a synergistic accumulation may be an overly simplistic explanation for the pattern in Panel A. For instance, it is possible that individuals from disadvantaged early-life SER do not actually benefit less from all incremental gains in adult SER. Instead, it may be that they are simply hurt less by very low adult SER, akin to a ceiling effect. In other words, mortality risks may rise only so high under existing social and epidemiological contexts. Panel B depicts this scenario with unparallel slopes among low-educated adults (in this example, less than a high school diploma). Thus, more compelling support for synergistic accumulation would come from the pattern reflected in Panel C, which more clearly depicts accelerated gains from adult SER among those from advantaged childhoods through unparallel slopes among high-educated adults (in this example, more than a high school diploma). Panels B and C make it clear that teasing out the accumulation hypotheses requires more than the conventional 2 x 2 matrix of childhood SER (low, high) and adult SER (low, high).

Hypothesis 4: Catch-Up Accumulation of Life Course SER

This final hypothesis asserts that the benefits of adult SER on functioning and mortality will be greater for individuals raised with disadvantaged, rather than advantaged, early-life SER. The underlying theory is that, because adults from advantaged early contexts are already operating near optimal levels of health, adults from disadvantaged early contexts have the most to gain from additional health inputs. Panel D

in Figure 4.1 illustrates how scholars typically envision this hypothesis in terms of mortality risk: the slope of mortality risk reduction with increasing adult SER is steepest for individuals raised in disadvantaged childhood SER.

In support, one U.S. study found that high adult SER had larger benefits for self-rated health, functional limitations, depressive symptoms, self-rated memory, and cognitive functioning, but not chronic conditions, among adults from low childhood SER than those from high childhood SER (Luo and Waite 2005). Empirical support also comes from Norway for all-cause and cardiovascular mortality for men and women, for accidental and violent deaths among men, and psychiatric mortality among women (Claussen, Davey Smith, and Thelle 2003).

However, a catch-up accumulation hypothesis may be too simplistic of an explanation for the pattern in Panel D. One possibility is that individuals from disadvantaged early-life SER do not actually benefit more from all incremental gains in adult SER. Instead, it may be that individuals from disadvantaged early contexts are simply hurt most pronouncedly by adverse adult SER, akin to a vulnerability effect. Panel E reflects this scenario with the steep and unparallel slope among low-educated adults. In support, a study of British males found that having a low income in adulthood or being employed in a manual occupation elevated the risk of coronary heart disease substantially more for men who were thin at birth than those who were not (Barker et al. 2001). Thus, more compelling support for catch-up accumulation would come from Panel F, which depicts accelerated gains from adult SER among those from disadvantaged childhoods through unparallel slopes among high-educated adults.

Given the mixed evidence regarding how exposures combine across the life course to shape adult functioning and mortality, further work is needed to systematically investigate the issue. The inconsistencies likely result from a multitude of factors such as the selected indicators of certain exposures (e.g., various indicators of socioeconomic resources), various adult age ranges examined, inattention to potential race and gender differences, various health outcomes and their unique etiologies, as well as overarching structural factors including epidemiological and sociopolitical context. Another major challenge for addressing this issue is data limitations. Specifically, interactions between childhood and adulthood SER are particularly difficult to estimate within datasets where there is limited intergenerational mobility.

This study addresses many of the gaps outlined above in our understanding of whether and how life course exposure to SER combines to predict functional limitations and all-cause mortality risk. Specifically, this study addresses the following questions for non-Hispanic white and black men and women 50 years of age and older in the United States.

1. To what extent are early-life SER and adult SER associated with functional limitations and all-cause mortality risk in later life?
2. Do early-life and adult SER exposures accumulate additively or interactively on later-life functioning and all-cause mortality risk? If they accumulate interactively, does the data support a Synergistic Accumulation or a Catch-up Accumulation? Does the data further support biological imprint and/or social

pathway hypotheses in explaining the links between early SER and these two outcomes?

DATA AND METHODS

Data

The data come from the 1992 through 2008 waves of the Health and Retirement Study (HRS). The HRS is a longitudinal household survey designed to study retirement processes, economic well-being, and health among U.S. adults 50 years of age and older (HRS 2008). This version of the HRS contains 30,548 adults who are representative of all cohorts born between 1890 and 1953 and their spouses. The present study uses the RAND HRS Version J Data File, which is a cleaned and consolidated data file of all 1992 through 2008 survey waves, developed by the RAND Center for the Study of Aging and supported by the National Institute on Aging and the Social Security Administration (RAND 2010). One benefit of using the HRS for the present study is that its birth cohorts span more than 50 years during which intergenerational educational mobility was relatively common, owing in part to the secular increase in educational levels and the G.I. Bill of Rights. A sufficient degree of intergenerational mobility—both upwards and downwards—is necessary for statistically distinguishing additive from interactive associations between early-life and adult SER.

Sample

The analytic sample is based on a person-year file, which was created by aging adults by one year beginning with their first interview year and until their year of death, or until 2008 if they survived the follow-up period. The sample retains person-year records for U.S.-born, non-Hispanic white and non-Hispanic black adults 50 years of age and older. The sample excludes a small number of adults (N=7) who did not provide their educational attainment. The final sample for the mortality analysis contains 24,326 adults representing 271,421.5 person-years of exposure and 8,207 deaths during the follow-up period. Because the functional limitations questions were asked most consistently starting in the second wave of the HRS (i.e., 1994), data from the first wave are excluded for the analyses on functioning. Thus, the final sample for functioning contains 107,165 person-years of exposure. Note that the sample size for the functioning analysis is roughly one-half the size of that for the mortality analyses. This is because, in the mortality analysis, person-years of exposure are created for every year (or fraction year) that the adult was alive, while in the functional limitations analysis person-years are only created for each biannual survey wave (not years in between waves) to avoid imputing functioning scores between waves.

Vital Status Ascertainment

The HRS provides vital status information from two sources. One source is the HRS tracker file (Tracker 2008, Version 1.0). In this file, adults are classified as dead or alive at each interview wave based on information gathered during the interview process (e.g., if a spouse reported that a study member died since the last interview, the adult is

classified as deceased). Month and year of death are then ascertained through an exit interview with a knowledgeable individual, such as spouse. The second source is the National Death Index (NDI), which is a computerized database of all certified deaths in the United States since 1979. The HRS provided information to the NDI on adults whose vital status was unconfirmed or presumed dead. The NDI then assigned vital status and date of death information through a probabilistic matching algorithm (Lochner et al. 2008; National Center for Health Statistics 2009). For the present study, an adult is considered deceased if either source classified the adult as dead.

Functional Limitations

Beginning in wave two of the HRS, adults were asked whether they had any difficulty with (including inability to do) 11 different functions because of a health or memory problem, excluding difficulties that they expected to last less than three months. The functions included walking one block, walking several blocks, sitting for two hours, getting up from a chair after sitting for long periods, climbing several flights of stairs without resting, climbing one flight of stairs without resting, stooping/kneeling/crouching, lifting or carrying weights over 10 pounds, picking up a dime from a table, reaching arms above shoulder level, and pushing or pulling large objects. The measure used in this analysis is the count of the number of functions that the adult reported some degree of difficulty with or inability to do.

Education, Age, and Race

The main predictors include the education levels of the adults' mother and father, as well as the adults' educational attainment. Research in the United States has increasingly focused on educational attainment as the key indicator of socioeconomic resources. Compared with income and occupation, education is a more stable measure across age, it is available for men, women, and individuals outside of the labor force (particularly important for studies of older adults), and it is most closely associated with health behaviors (Mirowsky and Ross 2003; Winkleby et al. 1992).

Mothers' and fathers' education were included as two binary variables indicating whether each had at least eight years of education. This specification is used because this is the only level of detail available in a few survey waves. In some cases, adults did not provide one or both parents' education level. Consistent with other studies that found adults in the HRS who were missing information on their father's (mother's) education level were similar on other economic and health variables to adults who reported their father (mother) had less than eight years of education (Luo and Waite 2005; Montez and Hayward 2011), missing values for parents' education were imputed accordingly.

In the first stage of the analysis, adults' educational attainment is included as a single binary variable (less than a high school diploma or GED=1). In the second stage of the analysis, adults' educational attainment is expanded into three binary variables reflecting less than high school, a high school diploma or GED (omitted reference), or more than high school. Lastly, all analyses were adjusted for age and race/ethnicity. Age

is a time-varying, continuous variable spanning ages 50 years of age and older. Race/ethnicity is a binary indicator for non-Hispanic white (hereafter white) or non-Hispanic black (hereafter black).

Methods

Poisson regression models are estimated to examine how SER in early life (measured by mothers' and fathers' education) and adulthood (measured by own education) are associated with all-cause mortality risk and functional limitations. The Poisson models estimate the natural logarithm of the annual mortality rate (or count of functional limitations) as a linear function of the predictors, and in the mortality analyses they also account for the smaller exposure interval for person-year records tied to respondents' first HRS interview year and their year of death. All models were estimated with SAS (Version 9.2) and adjusted for the HRS sample weights.

For each of the four race-gender groups, the Poisson models examine: (a) the extent to which mothers' and fathers' education is associated with the two outcomes, (b) the extent to which the association is mediated by adults' educational attainment assuming the early-life and adult education exposures accumulative additively to predict the two outcomes, and (c) whether early-life and adult SER accumulative interactively on the two outcomes, based on statistical significance of the interaction coefficients, and model fit comparisons using the Bayesian Information Criterion (BIC). The test for an interactive accumulation is conducted in two stages. In the first stage, the interaction terms are defined by the conventional 2 x 2 matrix of parents' education (less than 8

years, 8 years or more) and adults' education (less than high school, high school or more). The second stage uses a 2 x 3 matrix by expanding adults' educational attainment (less than high school, high school diploma or GED, more than high school) in order to evaluate the synergistic and catch-up accumulation hypotheses.

RESULTS

Table 4.1 contains key demographic information for the analytic sample. Among this sample, approximately one-third of whites (33 percent of men, 36 percent of women) and two-thirds of blacks (66 percent of men, 65 percent of women) reported having a low-educated father, one of the two proxies for early-life SER. Reflecting the secular increase in education levels, only about one-fifth of whites (18 percent of men, 19 percent of women) and two-fifths of blacks (44 percent of men, 40 percent of women) reported having attained less than a high school diploma or GED. The table also shows a moderate amount of educational mobility—both upward and downwards—between fathers and the sample adults, which is particularly important for estimating interactions between early-life and adult SER on mortality risk and functioning. In total, the analytic sample for the mortality analysis contains 115,874.5 person-years of exposure with 3,913 deaths among men, and 155,547.0 person-years of exposure with 4,294 deaths among women. Table 4.1 also corroborates prior research by documenting that, among the 11 functional limitations inquired about within the HRS, women reported an average of 2.9 limitations compared with just 2.0 among men; and blacks reported more functional limitations than whites.

All-Cause Mortality Risk

Table 4.2 contains coefficients from nested Poisson regression models estimating the natural logarithm of the annual risk of death from early-life and adult SER using the 2 x 2 interaction term, while Table 4.3 replicates the models using the 2 x 3 term. The results are discussed below for each gender-race subgroup in turn.

In Table 4.2, for the combined group of white and black men, models 1a-c support the synergistic accumulation hypothesis. Specifically, model 1a shows that both fathers' and mothers' education were inversely related to the risk of death (for example, having a low-educated father increased the annual risk of death by exp(0.136), or 14.6 percent, net of mothers' education), and fathers' education was attenuated but remained statistically significant after adult education was included in model 1b. Model 1c reveals that early-life and adult SER actually "interacted" such that the decrease in the risk of death associated with higher adult educational attainment was shallower for men with low-educated fathers: a conclusion derived from the significant and negative interaction term (-0.159, $p<0.05$), the smaller BIC value in model 1c, and the left panel of Figure 4.2. The results for white men are similar: fathers' education remained significant once adult education was accounted for, and the negative interaction term in model 2c (-0.136, $p<0.10$) supports a synergistic accumulation, although the evidence is weak given the marginal significance of the term and the modest reduction in the BIC in model 2c. For black men, neither early-life nor adult SER significantly predicted the risk of death. The lack of significance may reflect a lack of statistical power (black men experienced just

658 deaths during the follow-up period), particularly because the magnitude of the coefficients in model 3c are comparable to those in models 1c and 2c, and because the pattern for black men in Figure 4.2 is similar to that for white men.

Table 4.3 shows a more stringent test of the accumulation hypotheses by estimating the 2 x 3 interaction term. For the combined group of white and black men, and for white men, the more complex interaction term lost its statistical significance, likely due to smaller cell sizes. However, we can still glean some insights into the nature of the accumulation from Figure 4.3. It suggests that the synergistic accumulation in Figure 4.2 may more accurately reflect a ceiling effect on mortality risk among very disadvantaged men (i.e., low-educated men with low-educated fathers). Note that the incremental reduction in mortality risk beyond a high school diploma is irrespective of fathers' education, such that there is a consistent gap between men with low- versus high-educated fathers. For black men, the substantively large interaction coefficients in Table 4.3 were not statistically significant so Figure 4.3 should be interpreted cautiously. The figure suggests both ceiling and floor effects. Black men from disadvantaged SER experienced little mortality risk reduction with their own educational attainment unless they achieved more than high school; while black men from advantaged SER experienced substantial reductions with their educational attainment until they achieved a high school diploma after which further reductions were negligible.

The patterns differed for women. In Table 4.2, among the combined group of white and black women only fathers' education significantly predicted the risk of death in model 4a and it remained significant after including adult educational attainment in

model 4b. However, while the interaction term in model 4c directionally indicates a synergistic accumulation, it is not significant. The results for white women in models 5a-c are similar. Among black women, mothers' not fathers' education marginally predicted the risk of death in model 6a, but it was attenuated to nonsignificance by the inclusion of educational attainment, which was a significant predictor. Taken together, Table 4.2 supports an additive accumulation of early-life and adult SER on mortality risk among white women, while only adult SER was important for black women. In Table 4.3, the 2 x 3 interaction coefficients remain nonsignificant, except for a marginally significant term among white women. As Figure 4.3 illustrates, among white women, risk reductions from own educational attainment up to a high school diploma were irrespective of fathers' education; however white women with high-educated fathers differentially reduced their mortality risk with their own post-secondary education compared with women with low-educated fathers—reflecting a synergistic accumulation. Among black women, early-life SER remained unrelated to the risk of death and adult educational attainment lost its statistical significance, the latter is likely due to a small number of deaths given the magnitude of the coefficient and the steep slopes in Figure 4.3

Functional Limitations

Table 4.4 contains coefficients from nested Poisson regression models estimating the natural logarithm of count of functional limitations from early-life and adult SER using the 2 x 2 interaction term, while Table 4.5 replicates the models using the 2 x 3 term.

In Table 4.4, for all three groups of men, the models support the synergistic accumulation hypothesis. For example, model 1a shows that both fathers' and mothers' education were significantly and inversely related to functional limitations (men with low-educated fathers had, on average, exp(0.263) or 1.3 more functional limitations than men with high-educated fathers, net of mothers' education). Unlike the mortality analyses where mothers' education was attenuated to nonsignificance once adults' education was accounted for, both fathers' and mothers' education were attenuated but remained statistically significant predictors of functioning in model 1b. Model 1c shows a strong interaction between fathers' education and own education such that being disadvantaged on both did not elevate functional limitations to the extent that would be expected based on an additive accumulation. The results for all three groups of women also support the synergistic accumulation hypothesis, which is clearly illustrated in Figure 4.4. The figure also shows the more pronounced pattern among women compared with men, which is largely due to the fact that there was a strong interaction between *both* parents' education and own education among women, whereas among men there was an interaction just between fathers' and own education.

Table 4.5 contains the model estimates when using the 2 x 3 interaction term and by in large, confirms the synergistic accumulation hypothesis. Because interpreting the four interaction terms in Table 4.5 for each subgroup can be cumbersome, Figure 4.5 is provided to facilitate interpretation. Table 4.5 and Figure 4.5 corroborate the findings using the 2 x 2 interaction term, and again reveal that the synergistic accumulation is pronounced among women and among black men. However, when estimating the 2 x 3

term, the accumulation pattern among white men appears additive rather than interactive, which is peculiar finding that requires further study.

DISCUSSION

This chapter examined whether and how exposure to socioeconomic resources during early-life (measured by fathers' and mothers' education levels) and adulthood (measured by own educational attainment) accumulated to predict functional limitations and all-cause mortality risk among U.S. adults 50 years of age and older. Specifically, it evaluated whether the functioning and longevity benefits of socioeconomic resources experienced in early life and adulthood accumulated additively such that the benefits of adult resources were irrespective of one's early-life resources, or interactively such that the benefits of adult resources were contingent upon the resources experienced in early life. In addressing these questions, the results also offer some insights into whether early-life socioeconomic environments impart a biological imprint on functioning and mortality, and/or whether they set in motion adult circumstances that shape these outcomes. The chapter goes beyond previous studies by: (a) investigating the accumulation by gender-race subgroups in line with an intersectionality approach, (b) analyzing functioning and mortality within the same sample, (c) including both fathers' and mothers' education, and (d) conducting a more refined test of the nature of the accumulation on functioning and mortality. The findings expand our understanding of life course epidemiology and how the social patterning of socioeconomic resources across the life course shapes adult functioning, longevity, and population health disparities.

The results revealed that the nature of the accumulation differed by health outcome, by race-gender subgroup, and by the statistical test of the accumulation of early-life and adult resources: tests that utilize a 2 x 2 interaction (two levels of parents' education x two levels of adult education) constrain the nature of the accumulation to be consistent across the entire education distribution, while a minimally complex 2 x 3 interaction (two levels of parents' education x three levels of adults' education) allows the accumulation to differ within portions of the distribution, as the data suggest. The findings clearly illustrate that analyses that rely on a simple 2 x 2 interaction to evaluate how exposures to socioeconomic resources across the life course accumulate to shape health-related outcomes can obscure underlying patterns and produce misleading conclusions.

In sum, the mortality analyses found the following patterns. Among white men and women, both early-life and adult socioeconomic resources significantly and independently predicted the risk of death, although their accumulation was non-additive throughout portions of the education distribution. Among white men with at least a high school diploma, those with low-educated fathers had a consistently higher risk of death than those with high-educated fathers (an additive accumulation), while the gap converged among men who achieved less than a high school diploma due to an apparent ceiling effect on mortality risk among low-educated men with low-educated fathers (an interactive accumulation). Among white women with at most a high school diploma there was no gap in the risk of death between women with low- versus high-educated fathers, while white women who achieved more than a high school diploma experienced

differential mortality reductions with increasing education if they had a high-educated father (a synergistic accumulation), although these differential gains were modest. Among black women, parents' education no longer predicted the risk of death once adult educational attainment was accounted for. Among black men, neither fathers' nor adults' education were statistically significant predictors. However, the magnitude of the coefficients was substantively large and the patterns indicated both ceiling and floor effects: black men with low-educated fathers experienced little mortality risk reduction with their own educational attainment unless they achieved more than a high school diploma; while black men with high-educated fathers experienced substantial reductions with their educational attainment until they achieved a high school diploma after which further reductions were negligible.

In sum, the functional limitations analysis found the following patterns. For white women, black women, and black men, exposures to early-life and adult socioeconomic resources accumulated synergistically on functional health. Unlike the mortality analyses, both fathers' and mothers' education were important and independent predictors of functioning. Among these three race-gender groups, adults with high-educated parents exhibited differential gains in functional health from their own educational attainment throughout the education distribution compared with adults with low-educated parents whose gains from their own education tapered off. The "tapering off" was particularly pronounced among black men and black women beyond a high school diploma. Among white men, exposures to early-life and adult socioeconomic resources accumulated additively.

It is abundantly clear from these findings that in order to advance our understanding of whether, how, and why exposures to socioeconomic (or other) resources accumulate across the life course to shape health and longevity, studies must recognize that the way these exposures accumulate markedly differs by health outcome (as differences in their etiology would suggest) and by demographic subgroup, not withstanding other important moderators such as time period, cohort, and sociopolitical context. In this study, functioning was much more closely hinged to life course socioeconomic resources than was mortality risk. Other studies have similarly found that morbidity and physical functioning are more closely tied to adult socioeconomic status than is mortality risk (e.g., Hummer, Benjamins, and Rogers 2004). Some scholars assert that—at least with respect to gender disparities—longevity differences primarily reflect inherent biological differences, while health differences primarily reflect socially-structured inequities (e.g., economic resources, psychosocial stressors) (Bird and Rieker 1999). Further, the findings here add to a small but growing number of studies showing race-gender differences in the relationship between early-life socioeconomic resources and adult health outcomes. For instance, a ten-year follow-up study of U.S. adults 18 to 30 years of age at baseline found that parents' education predicted cardiovascular risk scores more strongly among whites than blacks, and most strongly among white women and weakest among black men (Karlamangla et al. 2005). The authors speculated that the weak association among black men may reflect a host of environmental factors that limit the health-related returns to education including discrimination, residential segregation that limits job opportunities and access to healthy foods and recreational activities, and

fewer financial returns to each year of education among blacks than whites—factors that may be most pervasive in the lives of black men. Barriers such as these might explain why in the present study the reduction in the risk of death among black men appeared to plateau at higher levels of education.

Taken together, these findings provide support for both biological imprint and social pathway mechanisms linking early-life socioeconomic resources with later-life physical functioning for all four race-gender subgroups. For each subgroup, the strong inverse association between parents' education and functioning was attenuated (supporting a social pathway) but remained statistically significant (supporting a biological imprint) when adult educational attainment was accounted for. Interestingly, functioning was very responsive to mothers' education, independent of fathers' education; whereas mortality risk was primarily responsive to fathers' education. Further study is needed to understand whether and why the human capital characteristics of mothers and fathers differentially shape their offspring's health and longevity. The "early-life origins" literature has generally relied on fathers' education or occupation as the sole indicator of the early environment, which may have obscured the linkages and pathways through which early environments shape later-life health, particularly because the dearth of studies that incorporated mothers' education has found a strong influence of mothers' education (Case et al. 2005; Guralnik et al. 2006). Similarly, the data also support biological imprinting and social pathway mechanisms on mortality risk among whites. However, among blacks, the results are somewhat ambiguous. Parents' education had only a marginal association with mortality risk. If statistical power was not in

question, this would tip the balance toward a social pathway mechanism. It may be the case that parents' education is simply not a good measure of early-life socioeconomic environments among these cohorts of blacks.

A few limitations to this study should be mentioned. First, while educational attainment is a stable and powerful measure of socioeconomic resources (Mirowsky and Ross 2003; Winkleby et al. 1992), it is just one indicator of the overarching socioeconomic environment. Additional indicators such as parents' occupation, income, and childhood exposure to poverty, as well as adults' occupation, income, and wealth, also likely play a role in these adult health outcomes. Future research should consider integrating these additional measures to build a more holistic picture of the processes examined here. Another limitation that should be addressed in subsequent studies is a closer examination of cohort differences in these processes. To the extent that the returns to education have changed for the U.S. population overall, in addition to some evidence that the returns changed more for dramatically for some subgroups than others (Feldman et al. 1989; Jemal et al. 2008; Meara, Richards, and Cutler 2008; Montez et al. 2011; Pappas et al. 1993; Preston and Elo 1995), the life course accumulation patterns may also vary by cohorts. Future work should also seek to identify the key mediators of the associations and be cognizant of the possibility that the most salient mediators may differ between segments of the education distribution, as hinted by the differential slopes in the figures. Addressing these mediators could help answer questions such as why black men exhibited such a unique accumulation pattern for mortality risk. Specifically, why does "double advantage" (i.e., having high-educated parents and being high-educated

themselves) not translate into further gains in mortality reductions? Also, why were white men the only race-gender group who did not experience a synergistic accumulation of socioeconomic resources on physical functioning? Why did white women, but not white men, experience a synergistic accumulation of socioeconomic resources on mortality risk?

Conclusions

Exposure to socioeconomic resources in early life (measured by mothers' and fathers' education levels) and adulthood (measured by own education level) generally accumulated to predict physical functioning and all-cause mortality risk among U.S. adults 50 years of age and older; however, the nature of the accumulation differed dramatically by health outcome, by race-gender subgroup, and by location within the education distribution; the latter of which can only be evaluated by moving beyond the conventional statistical test of non-additive accumulation (i.e., a 2 x 2 interaction term). The health-related benefits of socioeconomic resources do not accumulate similarly for all demographic subgroups at all locations within the education distribution. Thus, future studies of the life epidemiology of adult functioning and mortality must consider the nuanced nature of the accumulation and the etiology of each outcome.

Figure 4.1: Hypothesized Interactive Accumulation of Early-Life and Adult Socioeconomic Resources on Mortality Risk

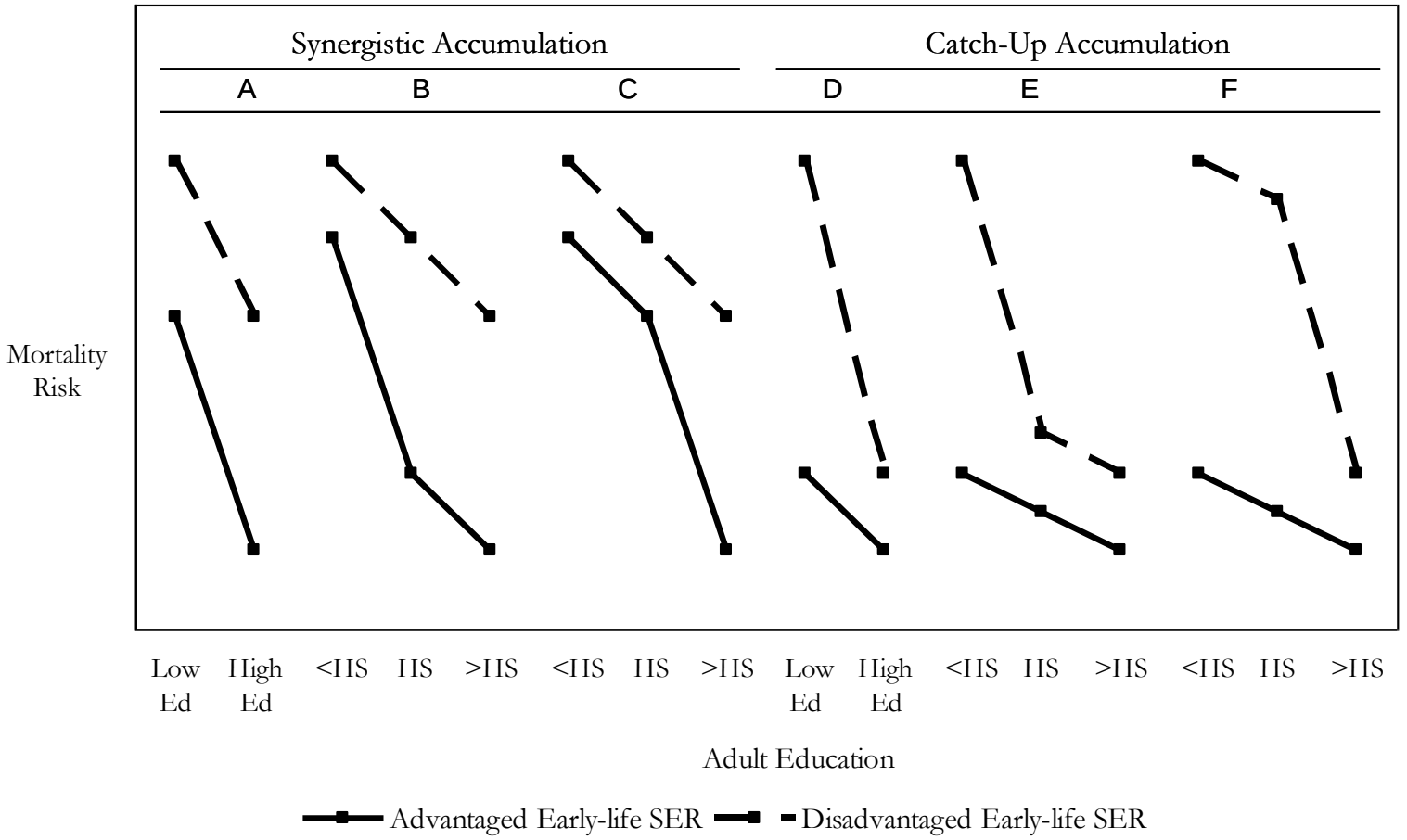


Table 4.1: Distribution of Parents' and Own Educational Attainment, Deaths, and Functional Limitations among Men and Women

	Men			Women		
	All	White	Black	All	White	Black
Age (years)	65.7	65.8	64.7	67.1	67.2	65.6
White (%)	91.0	---	---	89.5	---	---
Parents' Education (%)						
Father had < 8 years	35.7	32.7	65.5	38.6	35.6	65.1
Mother had < 8 years	26.9	24.7	48.8	30.1	27.7	50.4
Adult's Education (%)						
Less than HS	20.1	17.7	43.9	21.0	18.8	40.1
HS	33.5	34.0	28.1	40.3	41.5	30.2
More than HS	46.4	48.3	28.0	38.7	39.7	29.7
Fathers' & Adult's Education (%)						
< 8 years & less than HS	12.9	10.6	35.1	14.1	12.0	32.2
< 8 years & HS	12.8	12.3	16.9	16.0	15.6	19.3
< 8 years & more than HS	10.1	9.7	13.5	8.6	8.0	13.6
≥ 8 years & less than HS	7.2	7.0	8.9	6.9	6.8	7.9
≥ 8 years & HS	20.7	21.7	11.2	24.3	25.9	10.8
≥ 8 years & more than HS	36.4	38.6	14.5	30.1	31.7	16.2
Number of Deaths						
by Fathers' & Adult's Education						
< 8 years & less than HS	1,035	710	325	1,328	894	434
< 8 years & HS	610	507	103	733	598	135
< 8 years & more than HS	388	332	56	336	286	50
≥ 8 years & less than HS	447	367	80	463	362	101
≥ 8 years & HS	646	595	51	738	675	63
≥ 8 years & more than HS	787	744	43	696	642	54
Total	3,913	3,255	658	4,294	3,457	837
Person-Records	123,077	105,531	17,546	163,917	135,684	28,233
Person-Years	115,874.5	99,435.5	16,439	155,547.0	128,849.5	26,697.5
Death Rate (deaths / person-years)	0.034	0.033	0.040	0.028	0.027	0.031
Average Functional Limitations (SD)	2.0	2.0	2.6	2.9	2.9	3.7
	(2.7)	(2.7)	(2.5)	(2.9)	(3.0)	(2.5)
N (for mortality analyses; see notes)	46,081	39,518	6,563	61,084	50,306	10,778

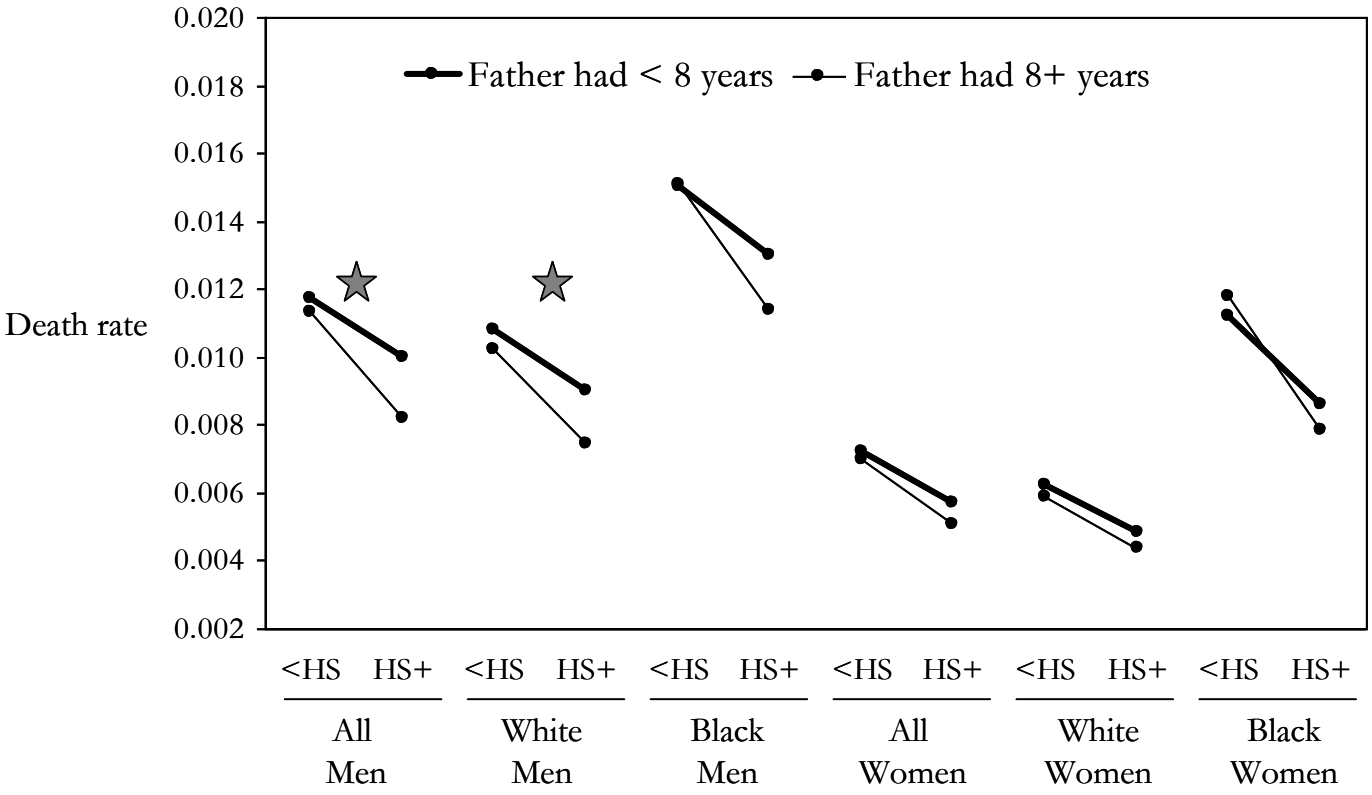
Notes: Distributions reflect person-records which are adjusted for the survey weights. SD means standard deviation. The range of functional limitations is from 0 to 11. The sample size is smaller for functional limitations than for deaths because functional limitation data are taken from survey waves 1994 onward, whereas death data are taken from waves 1992 onward.

Table 4.2: Poisson Regression Coefficients Predicting ln(Annual Death Rate) from a 2 x 2 Interaction Term

	White and Black			White			Black		
Men	1a	1b	1c	2a	2b	2c	3a	3b	3c
Intercept	-10.291**	-10.268**	-10.288**	-10.736**	-10.659**	-10.684**	-9.043**	-8.976**	-9.027**
Age	0.088**	0.086**	0.086**	0.090**	0.089**	0.089**	0.072**	0.070**	0.070**
White	-0.259**	-0.202**	-0.211**	---	---	---	---	---	---
Parent's Education									
Father had <8yr	0.136**	0.109**	0.196**	0.144**	0.115**	0.189**	0.032	0.016	0.136
Mother had <8yr	0.103*	0.066		0.098*	0.060		0.132	0.097	
Own Education (HS+)									
Less than HS		0.219**	0.322**		0.231**	0.317**		0.164	0.282
Interaction									
Father <8yr x LTHS			-0.159*			-0.136†			-0.141
BIC	31,393	31,375	31,373	27,934	27,917	27,916	3,469	3,477	3,477
Women	4a	4b	4c	5a	5b	5c	6a	6b	6c
Intercept	-11.445**	-11.407**	-11.414**	-12.086**	-11.997**	-11.995**	-9.386**	-9.275**	-9.336**
Age	0.098**	0.096**	0.096**	0.103**	0.101**	0.101**	0.072**	0.069**	0.069**
White	-0.265**	-0.209**	-0.211**	---	---	---	---	---	---
Parent's Education									
Father had <8yr	0.116**	0.084*	0.113**	0.131**	0.099*	0.103*	-0.003	-0.035	0.094
Mother had <8yr	0.063	0.002		0.041	-0.021		0.195†	0.127	
Own Education (HS+)									
Less than HS		0.263**	0.316**		0.268**	0.293**		0.280**	0.411*
Interaction									
Father <8yr x LTHS			-0.084			-0.047			-0.147
BIC	33,785	33,751	33,750	29,549	29,518	29,518	4,215	4,218	4,219

**p<0.01; *p<0.05; † p<0.10

Figure 4.2: Death Rate at Age 65 Estimated from Poisson Regression Models in Table 4.2

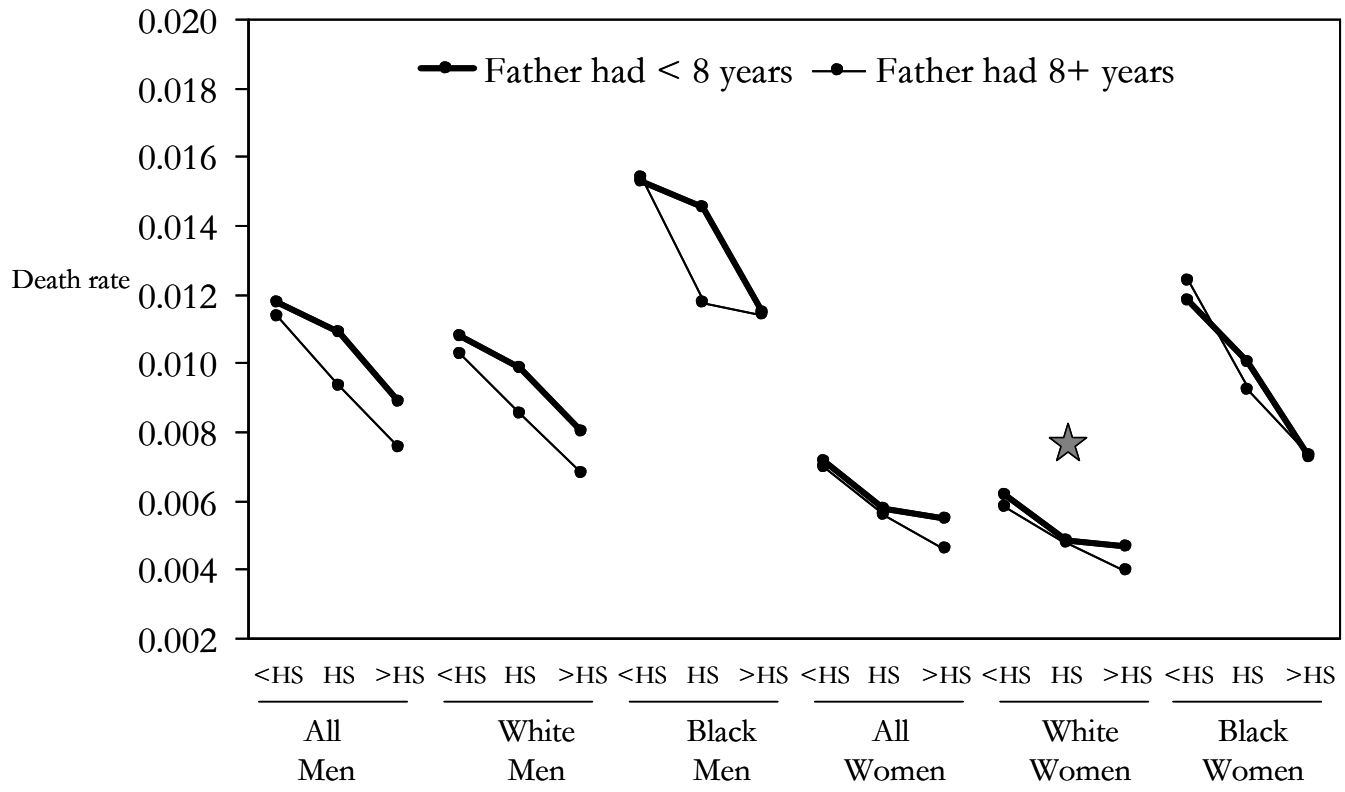


Notes: Stars indicate a statistically significant interaction.

Table 4.3: Poisson Regression Coefficients Predicting ln(Annual Death Rate) from a 2 x 3 Interaction Term

	White and Black			White			Black		
Men	1a	1b	1c	2a	2b	2c	3a	3b	3c
Intercept	-10.291**	-10.147**	-10.162**	-10.736**	-10.536**	-10.552**	-9.043**	-8.884**	-8.994**
Age	0.088**	0.086**	0.086**	0.090**	0.089**	0.089**	0.072**	0.070**	0.070**
White	-0.259**	-0.199**	-0.206**	---	---	---	---	---	---
Parent's Education									
Father had <8yr	0.136**	0.093*	0.155**	0.144**	0.099*	0.145*	0.032	0.008	0.213
Mother had <8yr	0.103*	0.050		0.098*	0.042		0.132	0.093	
Own Education (HS)									
Less than HS		0.117**	0.194**		0.125**	0.185**		0.099	0.268
More than HS		-0.215**	-0.216**		-0.218**	-0.222**		-0.157	-0.030
Interaction									
Father <8yr x LTHS			-0.117			-0.092			-0.218
Father <8yr x MTHS			0.012			0.015			-0.209
BIC	31,393	31,363	31,373	27,934	27,905	27,916	3,469	3,486	3,496
Women	4a	4b	4c	5a	5b	5c	6a	6b	6c
Intercept	-11.445**	-11.345**	-11.326**	-12.086**	-11.944**	-11.908**	-9.386**	-9.100**	-9.170**
Age	0.098**	0.097**	0.096**	0.103**	0.101**	0.101**	0.072**	0.068**	0.069**
White	-0.265**	-0.210**	-0.211**	---	---	---	---	---	---
Parent's Education									
Father had <8yr	0.116**	0.071†	0.037	0.131**	0.085†	0.015	-0.003	-0.050	0.083
Mother had <8yr	0.063	-0.009		0.041	-0.032		0.195†	0.109	
Own Education (HS)									
Less than HS		0.208**	0.220**		0.213**	0.192**		0.182	0.296
More than HS		-0.141**	-0.185**		-0.140**	-0.193**		-0.280†	-0.233
Interaction									
Father <8yr x LTHS			-0.008			0.041			-0.134
Father <8yr x MTHS			0.127			0.156†			-0.089
BIC	33,785	33,752	33,762	29,549	29,520	29,530	4,215	4,225	4,236

Figure 4.3: Death Rate at Age 65 Estimated from Poisson Regression Models in Table 4.3



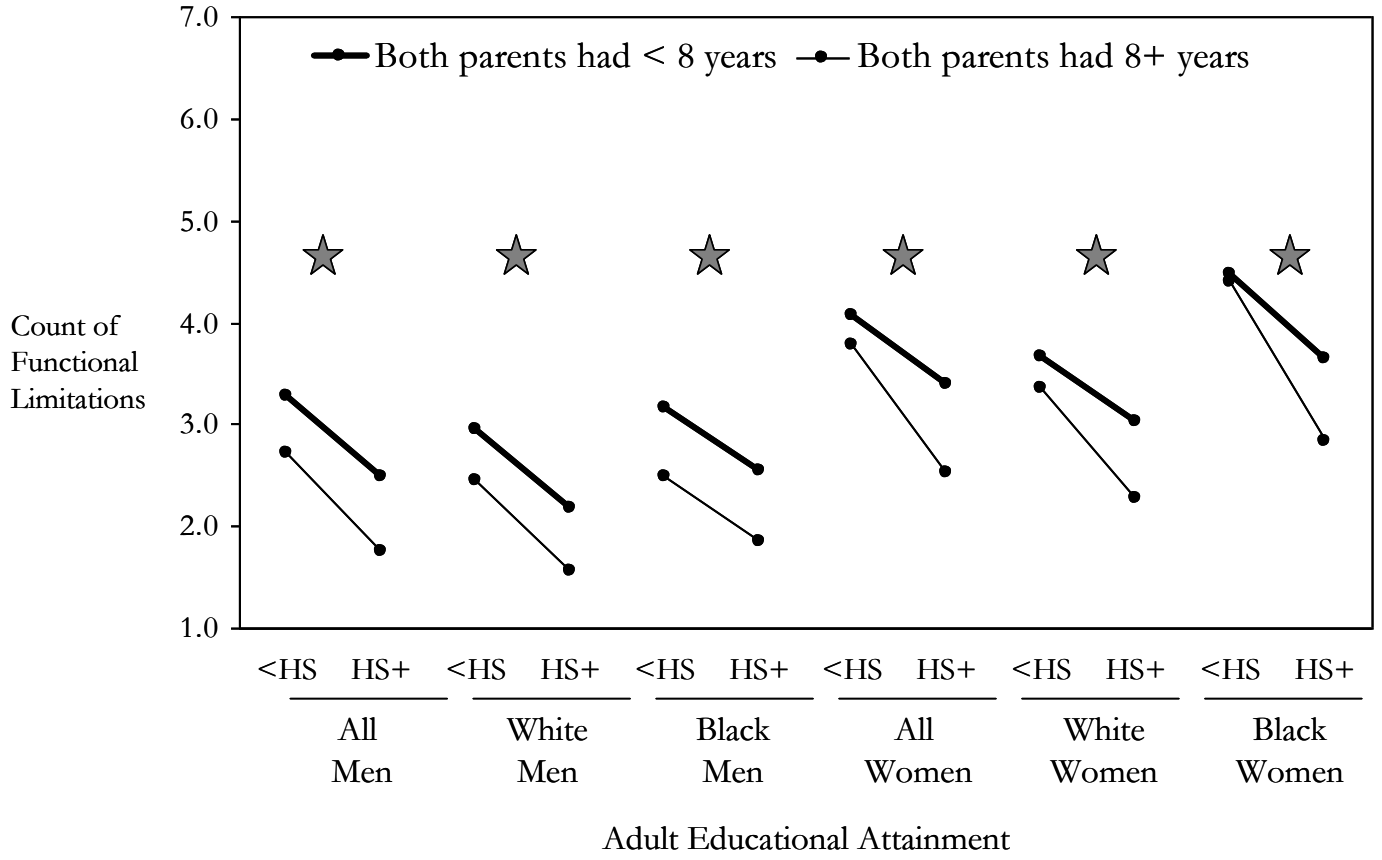
Notes: Stars indicate a statistically significant interaction.

Table 4.4: Poisson Regression Coefficients Predicting ln(Count of Functional Limitations) from a 2 x 2 Interaction Term

	White and Black			White			Black		
Men	1a	1b	1c	2a	2b	2c	3a	3b	3c
Intercept	-0.802**	-0.755**	-0.753**	-1.037**	-0.922**	-0.923**	-0.270**	-0.150*	-0.168**
Age	0.023**	0.021**	0.021**	0.024**	0.022**	0.021**	0.015**	0.012**	0.012**
White	-0.172**	-0.098**	-0.102**	---	---	---	---	---	---
Parent's Education									
Father had <8yr	0.263**	0.221**	0.254**	0.263**	0.216**	0.245**	0.237**	0.206**	0.241**
Mother had <8yr	0.136**	0.077**	0.092**	0.137**	0.079**	0.092**	0.141**	0.084**	0.079*
Own Education (HS+)									
Less than HS		0.348**	0.440**		0.371**	0.449**		0.236**	0.301**
Interaction									
Father <8yr x LTHS			-0.133**			-0.116**			-0.095†
Mother <8yr x LTHS			-0.030†			-0.029			0.010
BIC	222,645	220,839	220,765	198,835	197,064	197,021	23,748	23,637	23,650
Women	4a	4b	4c	5a	5b	5c	6a	6b	6c
Intercept	-0.181**	-0.146**	-0.157**	-0.469**	-0.404**	-0.414**	0.418**	0.559**	0.520**
Age	0.020**	0.018**	0.018**	0.021**	0.020**	0.019**	0.011**	0.008**	0.008**
White	-0.206**	-0.164**	-0.167**	---	---	---	---	---	---
Parent's Education									
Father had <8yr	0.207**	0.177**	0.200**	0.213**	0.182**	0.204**	0.140**	0.107**	0.122**
Mother had <8yr	0.108**	0.051**	0.092**	0.105**	0.049**	0.082**	0.126**	0.056**	0.129**
Own Education (HS+)									
Less than HS		0.278**	0.399**		0.283**	0.389**		0.293**	0.439**
Interaction									
Father <8yr x LTHS			-0.104**			-0.102**			-0.061†
Mother <8yr x LTHS			-0.113**			-0.097**			-0.171**
BIC	301,741	299,713	299,468	266,007	264,261	264,098	35,491	35,120	35,089

**p<0.01; *p<0.05; † p<0.10

Figure 4.4: Count of Functional Limitations at Age 65 Estimated from Poisson Regression Models in Table 4.4



Notes: Stars indicate a statistically significant interaction.

Table 4.5: Poisson Regression Coefficients Predicting ln(Count of Functional Limitations) from a 2 x 3 Interaction Term

	White and Black			White			Black		
Men	1a	1b	1c	2a	2b	2c	3a	3b	3c
Intercept	-0.802**	-0.547**	-0.533**	-1.037**	-0.710**	-0.702**	-0.270**	0.032	0.088
Age	0.023**	0.020**	0.020**	0.024**	0.021**	0.021**	0.015**	0.012**	0.012**
White	-0.172**	-0.094**	-0.096**	---	---	---	---	---	---
Parent's Education									
Father had <8yr	0.263**	0.188**	0.176**	0.263**	0.183**	0.159**	0.237**	0.189**	0.252**
Mother had <8yr	0.136**	0.055**	0.055**	0.137**	0.056**	0.081**	0.141**	0.067**	-0.179**
Own Education (HS)									
Less than HS		0.208**	0.243**		0.229**	0.257**		0.116**	0.078†
More than HS		-0.302**	-0.325**		-0.301**	-0.315**		-0.288**	-0.428**
Interaction									
Father <8yr x LTHS			-0.055**			-0.030			-0.106†
Father <8yr x MTHS			0.078**			0.096**			-0.093
Mother <8yr x LTHS			0.008			-0.017			0.268**
Mother <8yr x MTHS			0.007			-0.059**			0.569**
BIC	222,645	229,345	229,332	198,835	195,699	195,702	23,748	23,537	23,488

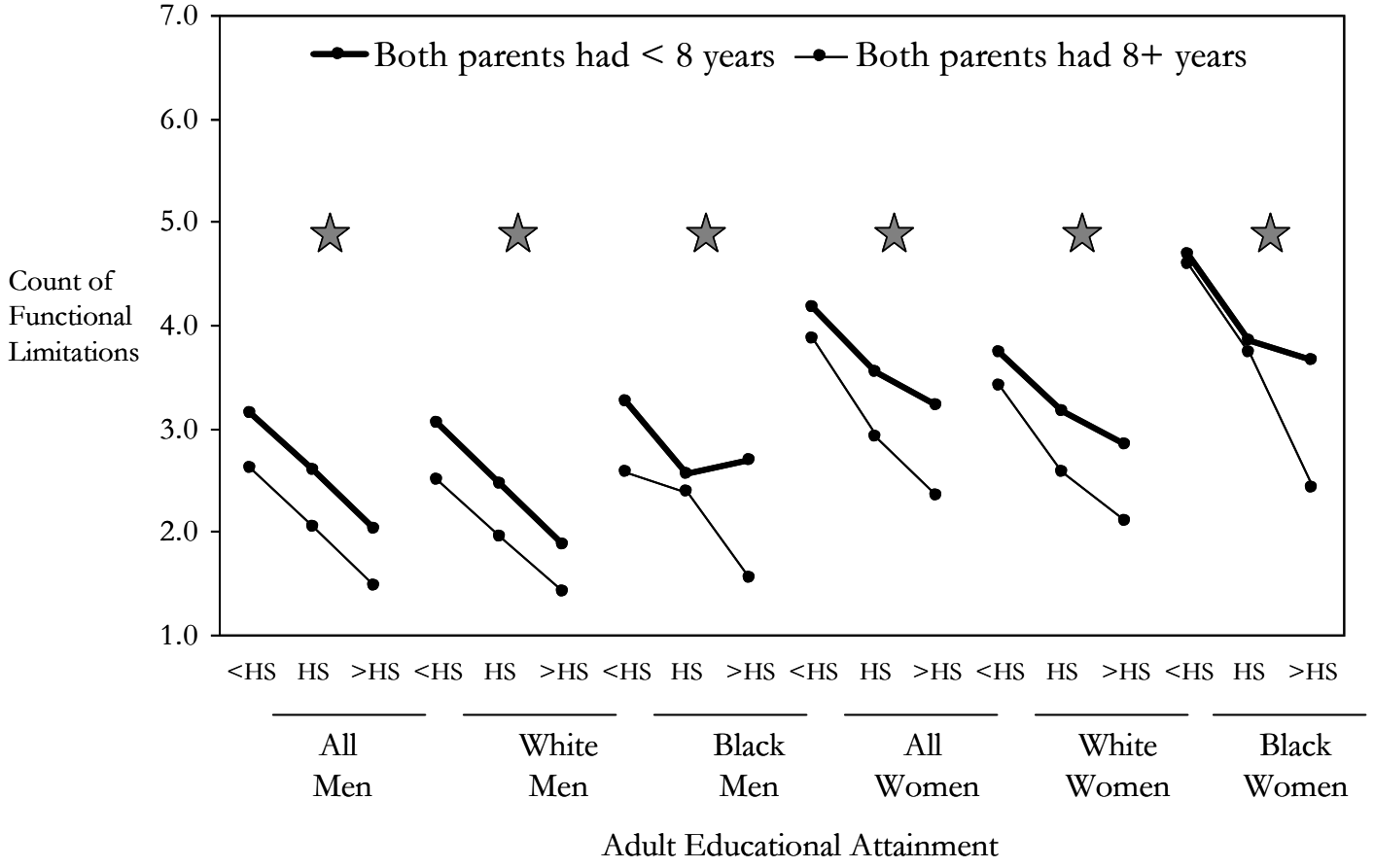
Continued on next page.

Table 4.5 continued.

	White and Black			White			Black		
Women	4a	4b	4c	5a	5b	5c	6a	6b	6c
Intercept	-0.181**	-0.014	-0.014	-0.469**	-0.285**	-0.286**	0.418**	0.743**	0.799**
Age	0.020**	0.018**	0.018**	0.021**	0.019**	0.019**	0.011**	0.007**	0.008**
White	-0.206**	-0.171**	-0.171**	---			---		
Parent's Education									
Father had <8yr	0.207**	0.154**	0.175**	0.213**	0.159**	0.184**	0.140**	0.085**	0.003
Mother had <8yr	0.108**	0.036**	0.022*	0.105**	0.032**	0.018†	0.126**	0.045**	0.024
Own Education (HS)									
Less than HS		0.201**	0.282**		0.209**	0.280**		0.184**	0.204**
More than HS		-0.199**	-0.218**		-0.193**	-0.204**		-0.261**	-0.433**
Interaction									
Father <8yr x LTHS			-0.080**			-0.081**			0.058
Father <8yr x MTHS			-0.010			-0.024			0.180**
Mother <8yr x LTHS			-0.042**			-0.031†			-0.064†
Mother <8yr x MTHS			0.133**			0.124**			0.206**
BIC	301,741	298,559	298,359	266,007	263,285	263,177	35,491	34,929	34,855

**p<0.01; *p<0.05; † p<0.10

Figure 4.5: Count of Functional Limitations at Age 65 Estimated from Poisson Regression Models in Table 4.5



Notes: Stars indicate a statistically significant interaction.

Chapter 5: Conclusions

The length and health-related quality of life differ dramatically between men and women in the United States and around the world. While contemporary American women can expect to live roughly five years longer than men, they can also expect to live a greater number of those years, and a greater percentage of their total years of life, functionally impaired. The consequences of this disparity are felt at both individual and population levels. At the individual level, the disproportionate burden experienced by older women not only hinders their quality of life, it impedes independent living and social engagement, elevates the risk of death, and can put strains on their adult children who must provide care and assistance. At a population level, the consequences play out in part through greater medical care and assistive needs of older women, which can be substantial, particularly as the U.S. population ages and women continue to outlive men.

For decades the scientific community has documented and sought to explain the disparity, but has largely fallen short of fully accounting for it. Most studies to date have focused on socially-structured inequities in adult circumstances and on differences in the prevalence of chronic conditions (e.g., arthritis) that precipitate poor functioning in adulthood. More recently, there has been increasing interest at the national level in unraveling the root causes by exploring potential causes further upstream in the life span. For instance, a 2001 report commissioned by the Institute of Medicine (Institute of Medicine 2001) advocated more resources and attention be devoted to explaining health

disparities between men and women: particularly to the potential role of endogenous sex differences and how they may interact with the environmental exposures across the entire life span—"from womb to tomb"—to produce the disparities. Referencing the 2001 Institute of Medicine report, the National Institute of Health issued a program announcement in 2010 for innovative, interdisciplinary research on the biological and social bases of health differences between men and women. Like the Institute of Medicine report, the announcement also explicitly sought research on whether and how these differences begin in the womb and accumulate across the life span.

Accordingly, this dissertation sought to advance our understanding of the gap in physical functioning and longevity between men and women by employing a biosocial, life course perspective. The main questions it addressed were: (a) are early-life experiences (particularly the socioeconomic environment) more consequential for women's than men's later-life functioning and longevity, (b) if early-life experiences are indeed more closely linked to women's than men's later-life functioning and longevity then which social and biological mechanisms explain the differential associations, and (c) how do socioeconomic exposures in early life and adulthood accumulate to predict functioning and longevity for women and men?

In summary, the findings suggest that certain early-life experiences (e.g., adverse socioeconomic environments) are indeed marginally more consequential for women's than men's later-life functioning and longevity, that the mechanisms linking early-life experiences and these outcomes are both social and biological in nature, and that the health-related consequences of socioeconomic environments accumulate across the life

course differently for women and men. Chapter Two revealed that women's functional limitations in midlife (45 to 74 years) were hinged to multiple early-life exposures, whereas men's were hinged to fewer exposures and less strongly so; that those exposures indirectly shaped functioning for men and women by setting in motion adult circumstances (e.g., educational attainment) that impact functioning; yet, certain early-life exposures (e.g., poverty) may also impart a direct, biological stamp on women's metabolic systems. Chapter Three focused one overarching early-life exposure—the socioeconomic environment—and found that it was important in staving off functional decline among adults 50 years and older—much more so than it was at helping adults recover from it or avoid death. There were few and subtle differences in how early-life socioeconomic resources shaped transitions across various states of functional ability for between women and men; although having fewer early resources disproportionately increased the likelihood of transitioning from a healthy state to functioning limitations among women than men, corroborating the findings in chapter two. Chapter Four also revealed a marginally greater vulnerability to adverse early-life socioeconomic adversities among women than men—particularly between white men, who enjoy better functioning with higher educational attainment irrespective of early-life socioeconomic exposures, and white women whose functioning gains plateau if they experienced early-life socioeconomic adversities. In sum, adverse socioeconomic circumstances in early-life have long-term consequences for later-life functioning and longevity for women and men, and the consequences appear marginally greater for women. While socially-structured inequities in the lives of men and women play a role, they do not fully explain

the gap; and these results, along with other cited research suggest that we should also explore inherent biological differences between men and women in contributing to persistent disparities in these health outcomes.

Ultimately, this research should reorient scholarly thinking about the origins of gender disparities in health and longevity in adulthood away from the conventional framework focused on adult circumstances, and toward a life course framework that recognizes the interplay between sex differences in biological vulnerability across the lifespan and gender differences in social experiences across the life course. It should also inform public health agendas by emphasizing programs aimed at prenatal and early childhood environments for men and women. The findings here underscore the importance of early-life environments in shaping the health of all Americans, and in setting the stage for health disparities in later life.

FUTURE RESEARCH

Mechanisms

One of the major findings in this dissertation is that the socioeconomic environment in childhood is a strong predictor of adult functioning and longevity. With the exception of chapter two, most of my research used parents' education as the sole indicator of that environment. The next step is to understand precisely why parents' education matters. Does it simply reflect material resources—for example, safe and hygienic housing, abundant and nutritious foods, and quality health care? Does it also reflect nonmaterial experiences in childhood—for example, regular meal and bed times,

cognitive stimulation in the home, parenting style, less parental stress and conflict, minimal pathogen exposure, neighborhood environments that facilitate social interaction and recreation, and quality schooling? Does it also reflect the prenatal environment—for example, are higher-educated mothers less likely to smoke during pregnancy and are they more likely to have access to quality prenatal care? Thus, my future research will integrate additional dimensions of the early-life environment to provide a more comprehensive picture and to pinpoint precisely which aspects of the socioeconomic environment explain the strong association between it and later-life health. Both MIDUS and the HRS contain a few additional dimensions, which may be helpful. However, I may need to turn to other data sources, such as The National Longitudinal Study of Adolescent Health, that are not typically used in aging research but that contain rich information on early-life environments.

Selection Effects

A common and valid concern when comparing health outcomes between older men and women is mortality selection. The concern is that the health gap between men and women is, to at least some degree, a byproduct of the least healthy men in a population not surviving long enough to become participants in surveys that commence in midlife. By extension, it is possible that the stronger associations between certain early-life exposures and later-life health among women compared with men may also reflect mortality selection. In other words, if men who experienced early-life adversities died prior to becoming eligible for the survey, then this limits the variation within the

male sample and thus masks statistical associations. This is a complicated issue to address. One potential way to address it is to utilize birth cohort studies, such as the 1946 National Birth Cohort Study in Britain. A second, but less direct approach, is to utilize surveys of younger respondents, such as The National Longitudinal Study of Adolescent Health, to circumvent age-related mortality selection concerns, although of course there are limitations on the degree to which the findings can be extracted to older respondents.

Biological Indicators

My future research will integrate biological indicators of multiple physiological systems including cardiovascular, immune, metabolic, and musculoskeletal. Both the MIDUS and HRS data now include measures of these biological indictors for a portion of their respondents. Integrating the biological indicators has multiple advantages. First, it will shed light on precisely how the physical and social exposures in early life and adulthood get "under the skin" to shape functioning and longevity of men and women. Second, they may reveal preclinical states of pathology that have not yet manifested and thus not reported by survey respondents. Third, using these indicators will also address potential concerns of differential reporting of health conditions between men and women.

Population Heterogeneity

I will also explore population heterogeneity in the linkages between early-life exposures and later-life functioning and longevity. Are there period and cohort differences in these processes? For instance, how do the linkages differ for adults who

were exposed to the Great Depression? How do the linkages differ for adults who were born before and after widespread implementation of the MMR (measles, mumps, rubella) vaccine? Are there differences in the linkages between race/ethnic groups, as the findings from the fourth chapter of this dissertation suggest? Do the processes vary by geographic region? In other words, do the strength and type of linkages differ between adults raised in urban versus rural regions?

Timing and Duration of Exposure

We also need a better understanding of the impact of timing and duration of early-life exposures on later-life functioning and longevity. For instance, does exposure to poverty in the first five years of life have more pronounced consequences than exposure during adolescence, and if so, why? What are the critical and sensitive periods for certain types of exposure? Further, are there dose-response effects such that each additional year of exposure to poverty deteriorates health or are there plateau effects? Subsequent studies need to consider these types of nuances in order to fully elucidate the pathways through which early exposures translate into later-life health and longevity.

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